# A CLINICAL STUDY OF INCIDENCE, RISK FACTORS, MATERNAL AND PERINATAL OUTCOME IN PREGNANCIES WITH ABRUPTIO PLACENTAE

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# CERTIFICATE

This is to certify that the dissertation titled "**A CLINICAL STUDY OF INCIDENCE, RISK FACTORS, MATERNAL AND PERINATAL OUTCOME IN PREGNANCIES WITH ABRUPTIO PLACENTAE"** is a bonafide work done by **Dr.S.SHEBA MATHAVI** in the Institute of Obstetrics and Gynaecology (Madras Medical College) Egmore, Chennai in partial fulfillment of the university rules and regulations for award of MS degree in Obstetrics and Gynaecology under my guidanceand supervision during the academic year 2012-2014.

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# DECLARATION

**I solemnly declare that this dissertation titled ""A CLINICAL STUDY OF INCIDENCE, RISK FACTORS, MATERNAL AND PERINATAL OUTCOME IN PREGNANCIES WITH ABRUPTIO PLACENTAE"** was done by me at Institute of Obstetrics and Gynaecology, Madras Medical College during the year 2012 - 2014 under the guidance and supervision of **Prof.DR.T.KRISHNAVENIM.D.,DGO. This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.S. Degree in Obstetrics and Gynaecology (Branch -II)**

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## ABSTRACT

AIMS AND OBJECTIVES:To study the incidence , risk factors,maternal ,perinatal morbidity and mortality of patients with placentalabruption.MATERIALS AND METHODS:A Prospective observational study;Studyperiod-oneyear,Studygroup:Patientsadmitted with clinical diagnoses of abruption with gestational age more than 28 weeks.,cases diagnosed retrospectively with retroplacentalclot.The maternal outcome in terms of anaemia,shock,DIC,renalfailure,postpartumhaemorrhageand death were evaluated.Perinatal outcome in terms of birth weight,Apgarscore,perinatal mortality were noted.RESULTS:Among 191 patients of antepartum haemorrhage the incidence of abruption was 0.76%(1 in132 deliveries.Maximum incidence was between 33-36 weeks of gestation and in multiparae.Hypertension and Anaemia are significant risk factors with ODDS ratio of 10.64 and 5.941 respectively.Coagulation failure:23.7%;Hypovolemic shock:37.62%;Renal failure:3.96%;Postpartum haemorrhage:34.65% and Couvelaire uterus:5.94% of patients.There were 2 maternal deaths in our study and the perinatal death rate was48%.CONCLUSION:Correction of anaemia and hypertension would help to reduce the incidence and early referrals to the teritiary care centres will decrease the maternal and perinatal mortality.

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# INTRODUCTION

The term abruptio placentae refers to a condition when haemorrhage occurs as a result of premature separation of normally situated placenta. It is one of the obstetric emergencies posing a threat tomaternal and fetal life.

The present study is done to know the incidence, risk factors of this disorder in our institution as well as the impact of this disorder on maternal and fetal outcome.

Though abruption is an obstetric emergency and a leading cause of perinatal morbidity and mortality the availability of diagnostic modalities like ultrasonagram has proved helpful in excluding other causes of ante partum haemorrhage like placenta previa and for timely management of the condition. Moreover the increased use of caesarean section and the better availability of haematological facilities has made a good impact in the recent studies.

All patients with diagnoses of abruption confirmed either clinically or by ultrasonagram or retrospectively following delivery were evaluated in our study and the required intervention was made to effect delivery either vaginally or by caesarean section by primary

clinical assessment of the patient and simultaneous treatment of haemodynamic instability or coagulation abnormality was carried out. The data generated from this study would help to improve maternal and fetal morbidity and mortality by planning prompt management of future cases of placental abruption.

# AIMS AND OBJECTIVES

To study the incidence and risk factors of patients presenting with placental abruption.

To study the outcome of patients with abruptio placentae in terms of maternal and perinatal morbidity and mortality.

# REVIEW OF LITERATURE

## GENERAL ASPECTS:

GENERAL ASPECTS: Third trimester bleeding is one of the most omnious complications of pregnancy and it complicates 3% of pregnancies. Third trimester obstetric haemorrhage seems to be one of the three leading causes of both maternal and perinatal morbidity and mortality. The obstetric causes of bleeding in third trimester being very hazardous has to be differentiated from the non obstetric causes.

Causes of third trimester bleeding

| OBSTETRIC | NON OBSTETRIC |
|---|---|
| 1.Bloody show | 1.Cervicitis |
| 2.placentaprevia | 2.Cervical polyp |
| 3.abruptio placenta | 3.Cervical erosion |
| 4.vasaprevia | 4.Vaginitis |
| 5.Disseminated intravascular Coagulation | 5.Genital tract trauma |
| 6.Uterine rupture | 6.Vulval,vaginalvaricosities |
| 7.Marginal sinus bleeding | |

**DEVELOPMENT OFTHE PLACENTA:**

The placenta is a remarkable organ performing many diverse functions including transport of gases and metabolites, immunological protection and production of steroid and protein harmones.

The zygote after repeated mitotic divisions gets converted in to a blastocyst. The outer layer of the blastocyst is called trophoblast and the inner cell mass is called embryoblast. The blastocyst after embedding in to the endometrium, the trophoblast rapidly proliferates and differentiates in to outer syncitiotrophoblast and an inner cytotrophoblast. The primary, secondary and tertiary villi are formed by the syncitiotrophoblasts and the intervillous space is formed by cytotrophoblasts. Until the end of sixteenth week the placenta grows in thickness and circumference due to the growth of the chorionic villi and expansion of the intervillousspace. After that there is little increase in thickness but it increases circumferentially until term. The implanted placenta by nature separates during the third stage of labor by a multiphasic process.

Abruptio placentae is the premature separation of a normally implanted placenta with haemorrhage in to the decidua basalis (Konje and Taylor2001)[1]. Antepartum haemorrhage complicates 2to5% of

pregnancies. Placenta previa and Abruptio placenta remain the two importantcauses of antepartum haemorrhage.

## HISTORICAL ASPECTS:

The term abruption in Latin means "breaking away" which describes the process by which the placental attachment to the uterus is disrupted by haemorrhage.

In 1609 Louis Bourgeois recognised the premature separation of placenta. DeLee and Coole in1848 coined the term ABRUPTIO PLACENTAE todenote sudden forcible separation of placenta from its normal site. In 1775, Edward Rigby of NORWICH made the first clinical differentiation between abruptio placentae and placenta previa. He also called abruptio placentae as accidental haemorrhage

## EPIDEMIOLOGY

The overall incidence of abruptio placentae varies from 0.5 to 1% (Ananth et al 1996; Baumannetal2000)[3]. Diagnoses is always clinical.

A pathologist often quotes a higher incidence of them have an unremarkable obstetric history. (Faye Petersen etal2006)[4].

The incidence varies due to variable diagnostic criteria as well as the increased recognition of milder forms of this disorder. Although various studies report a higher incidence of abruption in older women and those with increased parity (Konje andTaylor2001)[1]. There is conflicting evidence about the relationship of age and parity and abruption (Kramer etal 1997)[5].

**ETIOLOGY:**

The etiology is obscure but impaired placentation, placental insufficiency, uteroplacental underperfusion seem to be the underlying mechanisms

IMMUNOLOGICAL:

Immunological defects also may lead to an extensive inflammatory response with release of cytokines and result in shallow trophoblastic invasion, defective spiral artery remodeling, placental infarctions and thrombosis (Matthiesen et al1995)[6]

In placental abruption there is no suppression of cell mediated immunity and upregulation of humoral immunity which occurs in a normal pregnancy. This leads to exaggerated immune rejection of the fetus, activation of fetal monocytes and release of inflammatory

agents. HLA-G levels which avoid fetal rejection are decreased in abruption. If the signalling between trophoblastic and NK cells is poor it causes defective trophoblastic invasion and spiral artery remodeling which ultimately leads to dysfunctional placenta, thromboses, infarction and a generalised inflammation in which systemic endothelial dysfunction is a component. Hence placental abruption may be due to dysfunctional placenta caused by defective maternal immune response to paternal antigens. (Baumann et al 2000)

INFLAMMATION:

There is an increase of neutrophils and macrophages in placentae of women with abruption. There is increased production of TNF ALPHA and IL-BETA1 that increase the production of Matrix metalloproteinases by trophoblasts and other cell types. Increased premature production of Matrix metlloproteinases may destroy the extracellular matrix and cell to cell interactions causing premature placental detachment. (Ananth et al 2006a)

VASCULAR DISEASE:

A placental growth factor deficiency and sFlt-1 excess results from placental hypoxia associated defective remodeling of spiral

arteries. This defective remodeling leads to high resistance flow and finally rupture of decidual arteries.

## PATHOGENESIS AND PATHOPHYSIOLOGY OF ABRUPTIO PLACENTA:

Abruption is due to rupture of the decidual artery. The decidua is separated from the placenta by the accumulating blood. The collected blood when small in quantity may hardly produce any morbid pathological changes in the uterine wall or on the placenta. When the blood continues to dissect through the placental decidual interface it may lead to complete or near complete placental separation. After this the blood may escape through the potential space between chorion and decidua until it reaches the cervix. Blood may seep in to myometrium producing the so called COUVELAIRE UTERUS or reach the amniotic cavity producing blood stained amniotic fluid.

## COUVELAIRE UTERUS (UTEROPLACENTAL APOPLEXY):

It is a pathological condition first described by Couvelaire and is met with severe forms of abruption. There is massive intravasation of blood in to the myometrium up to the serous coat. The condition is diagnosed only on laparatomy. The uterus is of dark port wine colour

.It occurs initially at the cornu before spreading to other areas .Free blood may be present in the peritoneum or there may be a broad ligament haematoma. This myometrial haematoma rarely interferes with uterine contractions and hence it is not an indication per se for hysterectomy.

**HEPATIC AND RENAL CHANGES:**

In severe types of abruption placentae a fibrin knot which is a thrombotic lesion in the hepatic sinusoids has been described to be specific of abruptio placentae.

In renal changes oliguria and anuria may develop due to acute tubular necrosis in milder forms of abruption and renal cortical necrosis in severe forms of abruption.

**RETROPLACENTAL HAEMOTOMA[17]:**

It is a universal accompaniment of separation of placenta but is usually of little clinical significance. A fresh clot loosely adherent to the surface of a newly delivered placenta is usually seen if the placenta

is retained in utero some time after delivery but it is not the genuine retroplacental haematoma. If the placenta is not routinely seen many true retroplacental haematomas may be missed. Although many small lesions go unnoticed the large excavating retroplacental clot is readily apparent. Lesser degrees of indentations are produced by smaller lesions but the clot is usually firmly adherent to the maternal surface.

LARGE RETROPLACENTAL CLOT ON THE MATERNAL SURFACE OF PLACENTA

The colour of the haematoma is a function of the age. They lie between the basal plate and underlying endometrium and they are considered to be dissecting haemorrhages of the decidua. They have features similar to that of an intervillous thrombus histologically. Recurrent small haemorrhages may give a laminated appearance the oldest clot being near the placental surface.

Incidence of retroplacental clots is 5%.In known preeclamptics the associated abnormalities in decidual arterioles may be implicated. In otherwise normal pregnancies there is no clear indication of precipitating factors.

The clinical importance of retroplacental haematoma is two fold. Firstly the extent to which placenta remains deprived of its maternal blood supply clearly determines the survival of the fetus. However in otherwise normal pregnancies majority of these lesions have a little impact on fetal outcome. Secondly large retroplacental haematomas might result in consumptive coagulopathy in the mother.

## RISK FACTORS:

### AGE:

The incidence of abruption increases with maternal age. In the First and Second Trimester Evaluation of Risk trial(FASTER TRIAL) women older than 40 years were 2.3 times at more risk of abruption than those at35 years or younger. In a study by Bryan M Hibbard there is an increased incidence with maternal age and patients over 35 years are twice as prone to abruption as patients under 25 years. The unadjusted risk of abruptio placentae for women over the age of 35 years was 2.2 times the risk for women aged 19-34 years. (Williams et al)

### PARITY:

Although Pritchard and colleagues (1991) reported the incidence of abruption to be higher in women of great parity, Toohey and associates (1995) did not find this. HibbardBM and JEFFCOATE TNA[30] in their studies found an association between high parity and abruption

**NUTRITION:**

Folic acid deficiency seems to be an important factor in etiology of abruptio placentae. The possible association between folic acid deficiency and abruption is proved by bone marrow biopsy studies showing megaloblastic erythropoiesis. (Hourihana et al)

**HYPERTENSIVE DISORDERS OF PREGNANCY:**

The most common condition associated with abruption seems to be some form of hypertension (gestational hypertension, chronic hypertension, preeclampsia). Sibai and coworkers (1998) reported that 1.5% of pregnant women with chronic hypertension suffered placental abruption. Ananth and associates (2007) reported a2.4fold increased incidence of placental abruption with chronic hypertension and this was increased further if there was superimposed preeclampsia or IUGR. The severity of hypertension does not correlate with the incidence of abruption. (Witlin and colleagues Zetterstrom and coworkers 2005).

**PREMATURE RUPTURE OF MEMBRANES:**

There is an increased frequency of placental abruption in patients with early rupture of membranes. The incidence was 13% when premature rupture of membranes occured during gestational age

29-32 weeks. (Holmgren Paetal1997 Histological chorioamnionitis is associated with increased incidence of placental abruption and this association is dependent on its severity. (Nath et al 2007)[25]

**SMOKING AND COCAINE ABUSE:**

Smoking increases the overall risk of abruptioplacentae. According to a prospective cohort study the increase is by40% for each year of smoking prior to pregnancy. The rate of abruption has been reported to be13-35% in patients who abuse cocaine and seems to be dose dependent.

**TRAUMA:**

Blunt abdominal trauma or rapid decompression of uterus may cause shearing of placenta due to sudden stretching or contraction of uterine wall. Severe maternal trauma has been associated with a six fold risk of abruption.

EXTERNAL CEPHALIC VERSION:

When external cephalic version was a common practice abruption was a well known complication of it, especially when version was performed under anaesthesia. The incidence of abruption is said to be 2 to9% according to Savona Ventura (1986) who quotes 4 series of cases. The practice of version is now being revived and with

the addition of to colytic drugs the complication rates are reported to be low (Lancet Leader 1984)

**THROMBOPHILIAS:**

The association between placental abruption and the maternal thrombophilias supports the fact that abruption is a final acute clinical presentation of a chronic placental disease. (WENDY LKINZER etal)

**MULTIPLE PREGNANCY:**

The risk of placental abruption is increased 2-3 fold in multiple gestations (Salihu et al 2005[20], Ananth et al2001). In patients with twin gestation associated with placental abruption the risk of preterm birth or SGA is higher.

**PLACENTA PREVIA:**

About 10% women with placenta previa may have placenta previacoexistently. (Konje and taylor2001)[1]

**OTHERS:**

First trimester ultrasound examination showing a subchorionic or retroplacental haematomaincreases the subsequent placental abruption risk to 6-7 fold. (Balletal1996, NAGY et al 2003)

When the membranes rupture as in the case of poly hydramnios there is asudden decompression of the uterus .This leads to the reduction in uterine volume and a corresponding loss of surface area and as a result the placenta sheers off.

Abruption has been reported as a result of snakebite (Zugaib et al 1985) but has not been reported as a result of anticoagulant therapy. (Howell et al 1983[8])

Uterine malformations may lead to poor decidualisation and placentation. The contractility of a malformed uterus may lead to uncoordinated uterine action resulting in increased risk of placental abruption (Dabrashrafi et al1999).The risk of abruption is increased in patients with lower segment uterine scar due to impaired placental attachment.(Rasmussen et al1999[16]; Lydon Rochelle et al 2001)

**RECCURRENT PLACENTAL ABRUPTION:**

Ina women with a previous history of abruption there is a7-20 fold risk in a subsequent pregnancy (Karegard and Genser 1986[11], Ananth et al1996[3])

**PREDICTORS:**

**BIOCHEMICAL MARKERS:**

Unexplained second trimester elevation in MSAFP may be associated with subsequent adverse obstetric outcome including placental abruption. (Dugoff et al2005[12], Smith et al2006).This is due to chronic villitis and vascular thromboses or infarction (Salafia et al1988[13]).AFP levels >2.0MoM were detected in 17% of pregnancies with subsequent placental abruption (Finish study2002). Though MSAFP levels is a good marker for placental abruption it has not been used for this purpose widely in clinical practise. High levels of maternal serum beta hcg have been linked to placental abruption. (Liu et al1999).Decreased placental perfusion may lead to increased beta hcg.

Low levels of PAPP-A detected in the first trimester (29% at lowest 5th percentile and 43%at lowest 10th percentile) have been linked with placental abruption (Pilalis et al2007[14]). Proangiogenic placental growth factor (PIGF) and antiangiogenic soluble - fms like tyrosine kinase-1(sflt-1) are the regulators of angiogenesis in pregnancy. Increased levels of sflt1/PIG F ratios at21-32 weeks of gestation preceded subsequent placental abrubtion only in women who

has developed preeclampsia or PIH (Signnora et al 2006[29])

Fibronectin produced in the endothelial cell seems to be increased with placental abruption (Kanayama and Terao1992).

Levels of thrombomodulin which is a marker of endothelial cell damage may be increased with placental abruption (Magriples et al 1999).

D - dimer a by product of clot analysis may be used in early diagnoses of abruption (Nolan et al1993)

**Uterine artery flow measurement:**

High uterine artery pulsatility index at11-14 weeks or notching of the uterine artery waveform at 20-24 gestational weeks also may predict subsequent placental abruption (Pilalis et al2007[14]) but these methods have not been accepted universally in the prediction of abruption.

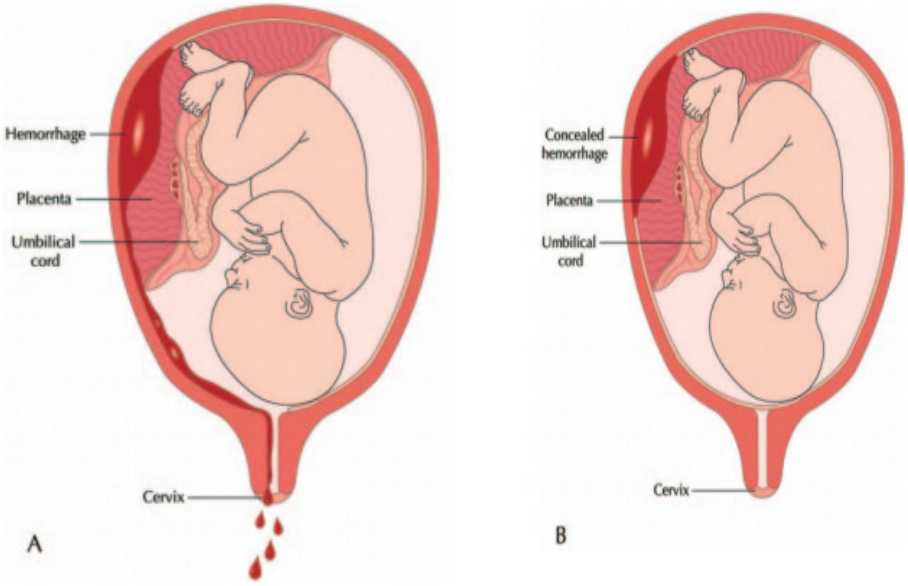
**FAMILY HISTORY:**

5% of women with abruption may have first degree relatives with abruption (Lindquist and Happach 2006[10]).

## CLINICAL PRESENTATION:

The clinical presentation varies from one patient to another. Vaginal bleeding, painabdomen, uterine tenderness, loss of fetal movements seem to be the classic symptoms. All these symptoms need not be present and asymptomatic presentation does not exclude placental abruption.

Placental abruption may be revealed or concealed. In the revealed type blood tracks between the membranes and the decidua escaping through the vagina. The less common concealed type occurs when blood collects behind the placenta without any external bleeding (Oyelese and Ananth2006[2])




A. Revealed type B. Concealed type

Four grades of placental abruption based on

**PAGE'S CLASSIFICATION:**

**Grade0** **:** An asymptomatic retroplacental clot seen after placental delivery.

**Grade1** **:** Present with vaginal bleeding and mild uterine tenderness.

**Grade2** **:** Vaginal bleeding may or may not be present but placental separation is significant enough to cause fetal compromise.

**Grade3** **:** Vaginal bleeding may be moderate or severe but there may be significant maternal complications along with late stage of fetal compromise or fetal death.

Geofrrey Sher (1978) proposed a clinical grading system

**Grade1** **:** Diagnoses is made retrospectively by seeing a retroplacental clot.

**Grade2** **:** Cases in which the fetus is alive. The retroplacental clot volume is usually 150 to 500gms.

**Grade3** **:** Features of Grade2 but fetal demise is confirmed. Grade 3 is further subdivided based on A) presence or B) absence of coagulopathy.

Vaginal bleeding is present in 70-80%of placental abruption though its amount poorly correlates with the degree of abruption. Uterine tendernss or pain is present in 66%. Hypertonic uterine contractions may be seen in 34% cases. Abdominal pain is less common in posterior placentations.

**DIFFERENTIAL DIAGNOSES IN CASES OF CONCEALED OR MIXED TYPES:**

1. Rupture uterus
2. Acutehydramnios
3. Rectus sheath haematoma
4. Appendicular or intestinal perforation
5. volvulus
6. Twisted ovarian tumor
7. Tonic uterine contraction
8. Red degeneration of fibroid.

**Points in favour of concealed abruption:**

1. Shock out of proportion to external bleeding
2. Unexplainedanaemia with tense uterus
3. presence of severe preeclampsia

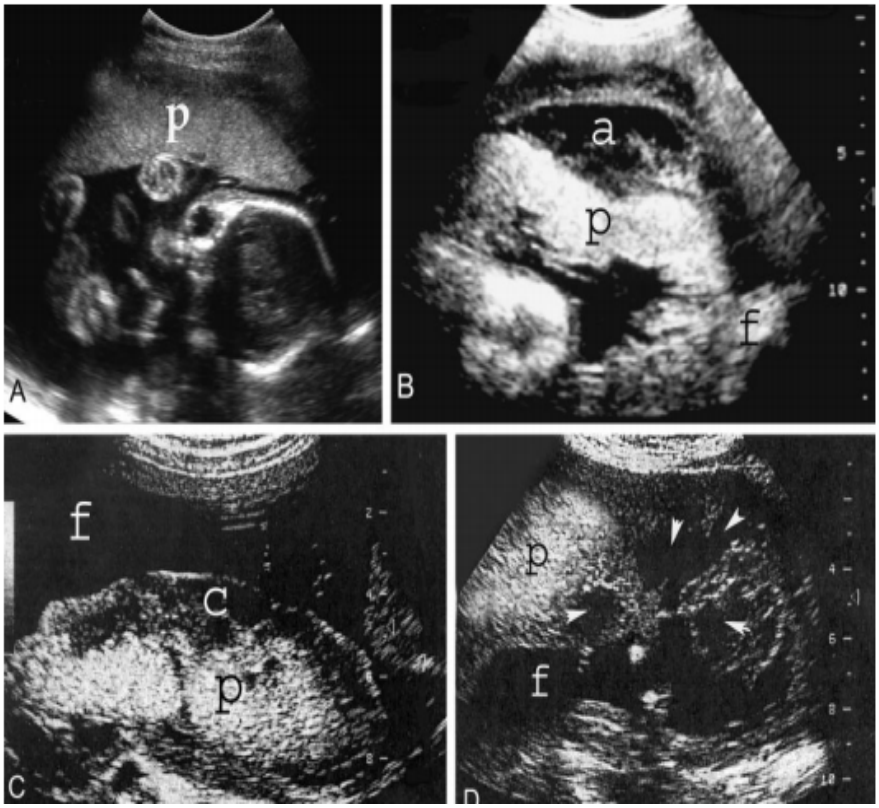
4. uterus may be tense tender
5. absent fetal heart
6. oliguria
7. coagulopathies

**ULTASONAGRAM IN ABRUPTION:**

Ultrasound abdomen is done

1. to rule out placenta previa
2. to reveal the state of fetus and degree of placental separation

In acute phase of abruption the haematoma appears hyperechoic to isoechoic when compared to placenta. On resolution the haematoma becomes hypoechoic within 1 week and sonoluscent within 2 weeks (Nyberget al1987).




A. Normal Placenta B, C Large Retro Placental Abruption D. Thickened Placenta with Heterogenous Appearance, Arrow Heads Show Areas of Haemorrhage.

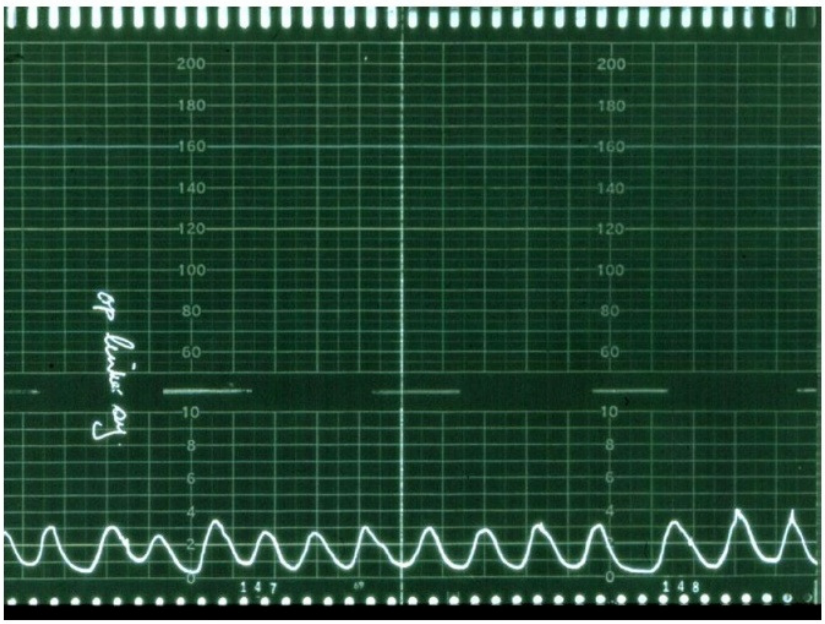
Although there is marked improvement in, the diagnoses of abruption was correct only in 25%. (Glantz and Purnell2002[31]). When a clot was visualised by USG, the positive predictive value of abruption was 88%.

Jello Sign:

The placenta may jiggle when sudden pressure is applied with transducer. However the ultrasound though inaccurate in diagnoses of abruption may be very helpful in excluding a placenta previa.

**CARDIOTOCOGRAPHIC CHANGES:**

Placental abruption may be associated with a variety of CTG patterns and this includes repetitive, late and variable decelerations, decreased beat to beat variability, bradycardia or sinusoidal fetal heart rate pattern (Oyelese andAnanth2006)[2]




CTG OF A DEAD FETUS WITH UTERUS SHOWING HYPERTONIC CONTRACTIONS (SAWTOOTH PATTERN)

**MATERNAL MORBIDITY:**

The maternal complications primarily depend on the severity of abruption. They include haemorhage and need for blood transfusions, coagulopathy, renal failure and hypovolemic shock, hysterectomy and less commonly maternal death. In cases of abruption severe enough to cause death of the fetus DIC sets in. Hypovolemiaor DIC may be a forerunner of acute renal failure.

HAEMORRHAGIC SHOCK**:**

There is significant loss of blood volume, either revealed vaginally or concealed within the uterus.

In pregnancy the increased blood volume and increased levels of coagulation factors such as fibrinogen and factor 7, 8 and 10 provide protection against haemorrhage. But when there is more than 25% loss of total blood volume, rapid haemodynamic deteroriation occurs.

When there is continuous bleeding and development of anaerobic metabolism with acidosis, there may be development of left ventricular failure and thereafter irreversible shock.

DISSEMINATED INTRAVASCULAR COAGULATION**:**

This is another common complication met with severe grades of abruptions associated with fetal death. In abruption placentae the release of procoagulant substances lead to release of tissue thromboplastins (from placental injury) in to the maternal circulation.

Due to the hypovolemia and hypoxia induced endothelial response there is white cell activation and production of proinflammatory cytokines and oxygen free radicals. These increase the oxidative stress and promote lipid peroxidation once antioxidant mechanisms are saturated. So there is loss of vascular integrity and hence increased vascular permeability.

The thromboplstins and the endothelial damage lead to widespread activation of coagulation cascade. If this goes unchecked, there is rapid consumption of coagulation factors and platelets with deposition of fibrin in microcirculation and thrombus formation in the placental surface. This leads to defibrination, thrombocytopenia and hemostatic failure. Disseminated intravascular coagulation also stimulates fibrinolysis and the resultant fibrin degradation products interferes with clot formation producing haemorrhage and also has a negative effect on cardiac function. The diagnoses of disseminated

intravascular coagulation is based on a combination of clinical and laboratory diagnoses. Bleeding can occur at venepuncture sites, gums, nose, rectum etc. Platelet counts and coagulation tests are abnormal .As more than 50% clotting factors must be consumed before the coagulation tests become abnormal, these tests become insensitive. As fibrinogen levels are increased in normal pregnancy, even low normal levels should be of concern. Fibrin degradation products including D-dimer are measured in the circulation and their abnormal levels help to confirm the presence of coagulopathy.

**RHESUS ISOIMMUNISATION**:

There may be a significant amount of fetomaternal haemorrhage. So it is advisable for all rhesus negative women with abruptio placentae to undergo a kleihauverbetke testto determine the correct dose of ANTI-D Immunoglobulin.

Maternal mortality however has decreased in the past few years due to increased maternal health care facilities. Also women with a history of placental abruption are less likely to become pregnant again when compared to control cohorts(Rasmussen et al1999)[16].

A study of long term effect of placental abruption on maternal health (Ray et al2005)[32] shows a risk of development of premature cardiovascular disease in 70% of these women. But the cause remains unclear.

**PERINATAL MORBIDITY AND MORTALITY:**

Perinatal morbidity includes low birth weight, preterm deliveries, birthasphyxia, stillbirth, intrauterinedeath, neonataldeaths. Here also the fetal survival depends on the grade of abruption as well as the gestational age.

If more than 50% of placental surface is involved fetal death invariably occurs. The perinatal mortality also depends on the prematurity of the fetus and the neonatal facilities available. Both spontaneous and iatrogenic preterm deliveries are common in placental abruption.

Fetal growth restriction is so well associated with abruption, that it could be taken as a single marker for risk of abruption (Ananth and Wilcox 2001)[24].Even congenital fetal malformations are approximately two times higher than in general population (Konje and Taylor2001[1]).The cause behind this remains unclear. Low Apgar

scores and cord blood pH values are due to maternal hypoxia (Spinillo et al 1993)[9].The risk of birth asphyxia is 3.7 fold. Long term risk of neonates born to mothers with placental abruption are development of cystic periventricular leukomalacia or intra ventricular haemorrhage. These neural defects increase with prematurity and low birth weight (Spinillo etal1993)[9]

**GENERAL MANAGEMENT:**

A rapid clinical assessment should bedone in women presenting with antepartum haemorrhage whether urgent intervention is required to manage maternal or fetal compromise. In women presenting with massive haemorrhage resuscitation should be started immediately since mother is the priority and should be stabilised prior to establishing the fetal condition. Meanwhile blood should be collected for investigations to identify complications.

**The investigations may include:**

The investigations may include

Blood Hb%

Platelet count

Leucocyte count

Peripheral blood smear

Blood for grouping and typing

Bleeding and clotting time

Clot observation and retraction time

Prothrombin time

Activated partial thromboplastin time

Serum fibrinogen level

Fibrin degradation product levels

D-dimer levels

Liver function tests

Renal function tests

Serum electrolytes

Arterial blood gas analysis

Kleivauher Betketest (if available)

**OBSTETRIC MANAGEMENT:**

The clinical presentation of placental abruption being variable, the obstetrical management is individualised on a case by case basis. The presentation, gestaional age and the degree of maternal and fetal compromise decides the management. In cases of abruption at or near term with a live fetus, prompt delivery is indicated. If there is fetal compromise, and delivery is not imminent, Caesarean section should

be performed. When the maternal and fetal conditions are reassuring vaginal delivery may be reasonable.

RUPTURING THE MEMBRANES AND HASTENING DELIVERY:

The main purose of doing an amniotomy is to hasten the onset of labour and by encouraging uterine contractions, to reduce uterine bleeding. It is effective and useful in most cases but seems to be dangerous in a few. If the uterus has become atonic as happens rarely, reduction of intrauterine pressure encourages further bleeding which fills the space. An intravenous infusion of oxytocin should always be started.

If patient is in established labor it may be allowed to progress in these conditions. On the other hand induction of labor may be planned if the labor has not been established. There should be close monitoring of both mother and fetus and when fetal heart rate becomes nonreassuring, caesarean section is indicated.

In patients with severe placental abruption resulting in intrauterine fetal death, if the mother is stable, it is reasonable to allow for a vaginal delivery in the absence of any obstetric indications. Labour usually progresses rapidly because of viguorous uterine

contractions and an amniotomy may speed up delivery. When labor does not progress rapidly or in case of obstetrical indications like cephalopelvic disproportion, a scarred uterus or a fetal malpresentation.

Caesarean delivery may be performed to avoid worsening of coagulopathy. But stabilisation of the patient and correction of any coagulation defects is very much essential during surgery. The patient should be closely monitored paying attention to vital signs, amount of blood loss, urine output. Uterus may remain hypotonic necessitating emergency hysterectomy. There should be a high index of suspicions of preeclampsia in patients with abruption because this may be masked by hypovolemia.

**MANAGEMENT OF MATERNAL COMPLICATIONS:**

Haemorrhagic shock is a major complication of abruption. Hypovolemia should be corrected early. Since there may be a greater degree of vasospasm blood pressure may not be a good guide to assess shock. So regardless of the patient's general condition atleast one litre of blood should be transfused. Central venous pressure is the best guide to monitor the patient .Haematocrit should be atleast 30% and urine output more than 30 ml per hour.

Disseminated intravascular coagulation is due to release of tissue thromboplastin in placental abruption leading to consumptive coagulopathy. Whole blood transfusion replenishes not only the fibrinogen but also other procoagulants. Fresh frozen plasma helps in the administration of fibrinogen, a necessary procoagulant. Prompt replacement of blood volume and coagulation factors helps a lot in the management of these coagulation disorders. There is no role of heparin or anticoagulants in disseminated intravascular coagulation caused by abruption.

Steps for immediate delivery of the fetus should be taken that would help to improve the haemostatic competence in vast majority of cases. Usually the coagulopathy settles immediately after delivery and

there is no clinically evident coagulopathy 12 after the delivery.

Renal failure in its early phase can be corrected in most cases of abruption by volume replacement .But however in its late phases the help of a nephrologist is needed.

**EXPECTANT MANAGEMENT:**

In case of preterm gestational ages when there is minimal abruption evidenced by ultrasound, there is a role of conservative management. But this should be individualised on a case to case basis.

These patients usually present with a small vaginal bleed and a localised area of uterine tenderness or an ultrasound evidence of minimal separation of placenta. In such circumstances there is something gained from allowing the pregnancy to continue but the decision to terminate will depend on the length of gestation, whether there was a previous episode the state of fetus and the extent of placental separation.

Abruption in a posteriorly situated placenta is dangerous because the only symptom being backache and also the placenta is out of reach.However innocuous the incidence of abruption seems to be

there is some damage to the integrity and function of placenta. And these patients need an extremely close monitoring because of a significant risk of fetal death at any period of time. Initial hospitalisation for further evaluation and fetal wellbeing is necessary and serial ultrasonagrams may be needed to evaluate progression or regression of abruption.

**PREVENTION OF ABRUPTIO PLACENTAE:**

It aims at prevention of risk factors likely to cause abruption, early detection of anaemia in the antenatal period and its correction and prompt identification and commencement of therapy to minimise grave complications namely shock, coagulopathy, renalfailure. Prevention of factors likely to cause placental separation are:

1. Early diagnoses and treatment of preeclampsia and other hypertensive disorders of pregnancy.
2. Avoidance of sudden rupture of membranes.
3. Avoidance of trauma especially forceful external cephalic version.
4. Regular intake of folic acid in early pregnancy.

## MANAGEMENT OF A SUBSEQUEUENT PREGNANCY:

These women are at a ten fold risk of having an abruption in the next pregnancy. They are also at increased risk of other pregnancy outcomes like preterm birth and preeclampsia. Some recommendations are possible in this women. Women who smoke tobacco or use cocaine should be encouraged to quit before next pregnancy. Hypertension should be controlled well before the next pregnancy. It is reasonable to treat women with inherited thrombophilias with thrombophylaxis although no clear benefit has been demonstrated. Because patients with abruption have an increased risk of impaired uteroplacental perfusion it is reasonable to do serial scans during the second half of subsequent pregnancy.

# MATERIALS AND METHODS

A prospective observational clinical study was carried out in our institution from Oct'12 to Sept'13 to determine the incidence, risk factors, maternal and perinatal outcome in patients presenting with abruption placentae. Study group consists of patients admitted to our labour room with clinical diagnosis of abruption and gestational age more than 28weeks. Cases diagnosed retrospectively with retroplacental clots were included in this study.

**INCLUSION CRITERIA:**

Pregnant women with gestational age more than 28 weeks with complaints of bleeding per vaginum.

Patients with retroplacental clots diagnosed retrospectively.

**EXCLUSION CRITERIA:**

Patients with atypical signs and symptoms were excluded from the study after their delivery if there was no clinical evidence of abruption.

A detailed history of the patient including the obstetric history was taken. A good clinical examination and an ultrasound examination

were done for the patients to arrive at a diagnoses and the patients diagnosed to have placental abruption were followed up for maternal and fetal outcome.

Risk factors for abruption were analysed by the following statistical methods in the patients presenting with abruption.

1. Chi square test,
2. Fisher exact test

Investigations including coagulation profile and the renal function tests were done to identify complications.

The grade of abruption presenting in the patient was identified and the severity was correlated with the maternal morbidity and mortality as well as fetal outcome in terms of intrauterine fetal death, stillbirths, neonatal admissions and neonatal deaths.

The fetal outcome was also measured on the basis of route of delivery and also the birth weight.

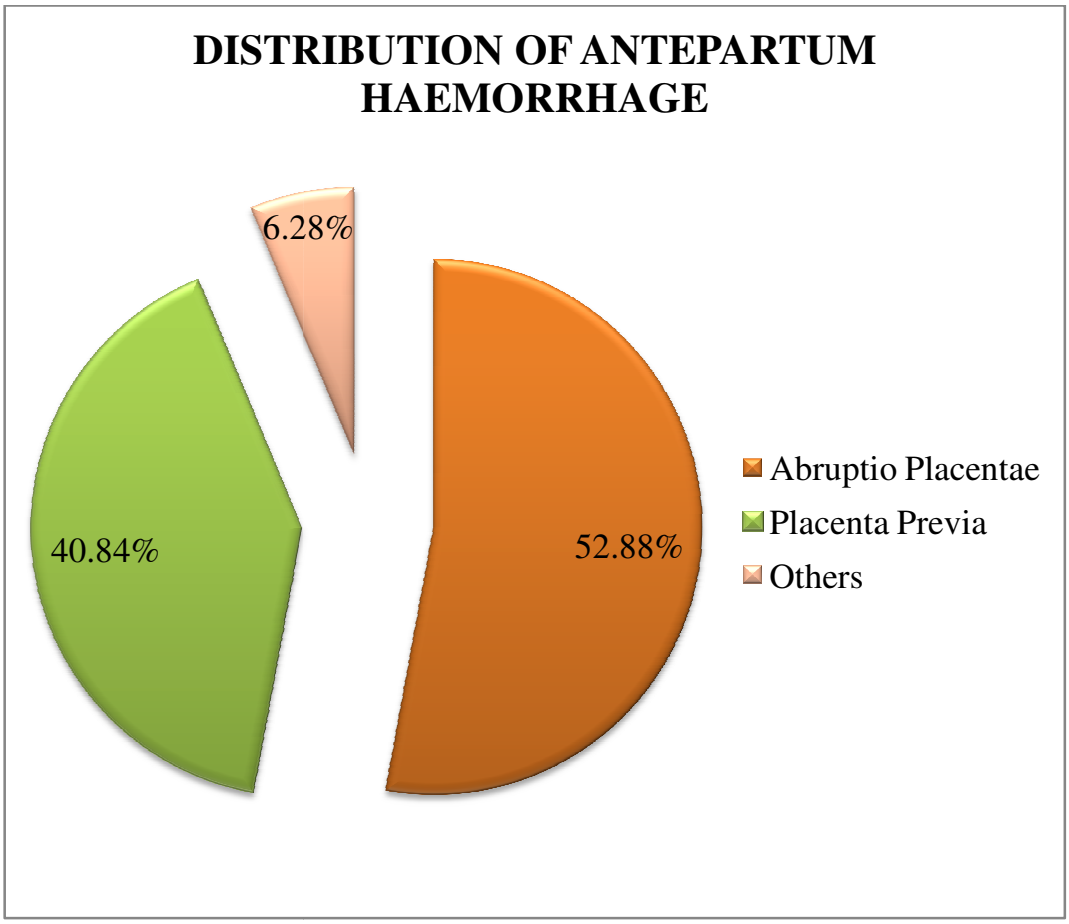
# RESULTS OF THE STUDY

## TABLE I

| Total No. of Deliveries in study period : 13239 | | |
|---|---|---|
| | **Incidence** | **%** |
| Antepartum Haemorrhage | 191 | 1.44% |
| Abruptio Placentae | 101 | 0.76% |
| Placenta Previa | 78 | 0.5% |
| Others | 12 | 0.09% |

The total number of deliveries in the study period was 13239. .Among these patients presenting with antepartum haemorrhage accounted for 191caseswith an incidence rate of 1.44%.The two main causes of antepartum haemorrhage are Placenta previa and placental abruption. In our study placental abruption accounted for 101 cases making up an incidence of 0.76% i.e,1 in132 deliveries.

The number of patients presenting with Placenta previa were78 with an incidence of 0.5%.The remaining patients of antepartum haemorrhage were12 and make up an incidence of0.09 %.



## TABLE II

## AGE DISTRIBUTION AMONG CASES OF PLACENTAL ABRUPTION

| Age Group | Incidence | % |
|---|---|---|
| < 20 years | 7 | 6.93% |
| 21-25 years | 52 | 51.48% |
| 25-30 years | 35 | 34.65% |
| 31-35 years | 6 | 5.94% |
| > 35 years | 1 | 0.99% |

When the age was analysed the highest incidence was among 21-25 years accounting for 52%.The youngest age at which abruption occurred was 18 years and the highest age was 38 years.

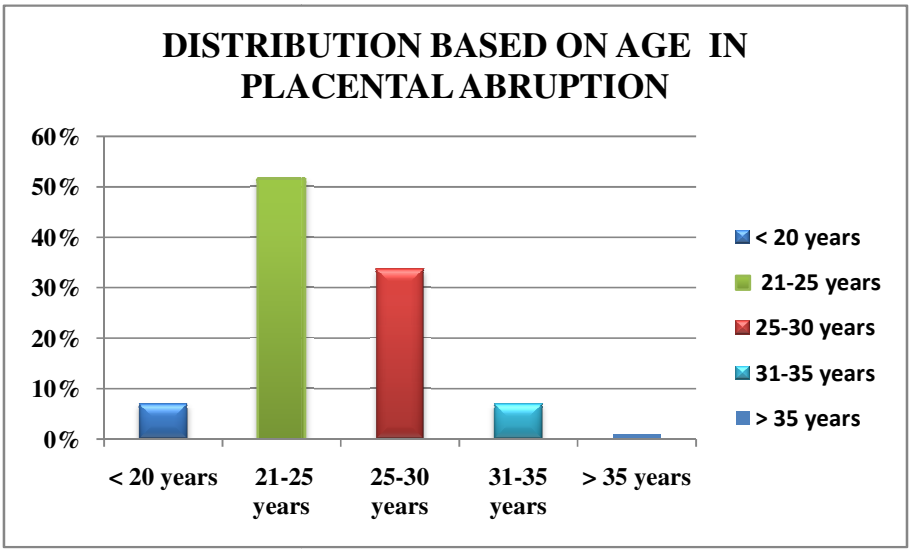
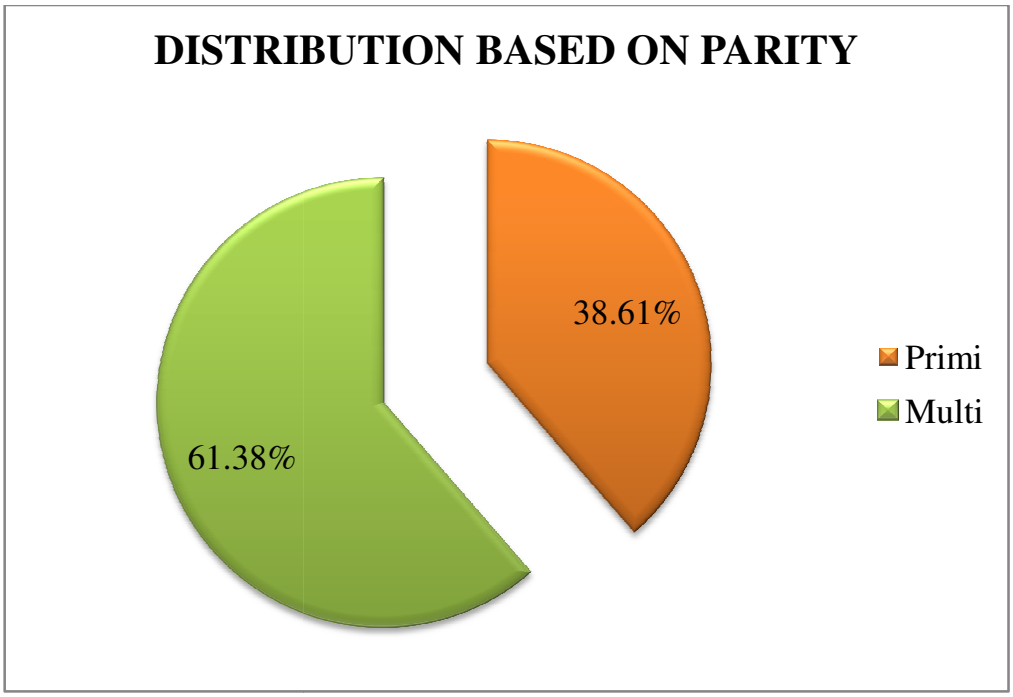


**TABLE III**

**DISTRIBUTION BASED ON PARITY (n=101 CASES)**

| | | |
|---|---|---|
| **Parity** | **Incidence** | **%** |
| Primi | 39 | 38.61% |
| Multi | 62 | 61.38% |

The incidence of abruption was seen to be highest among multipara.

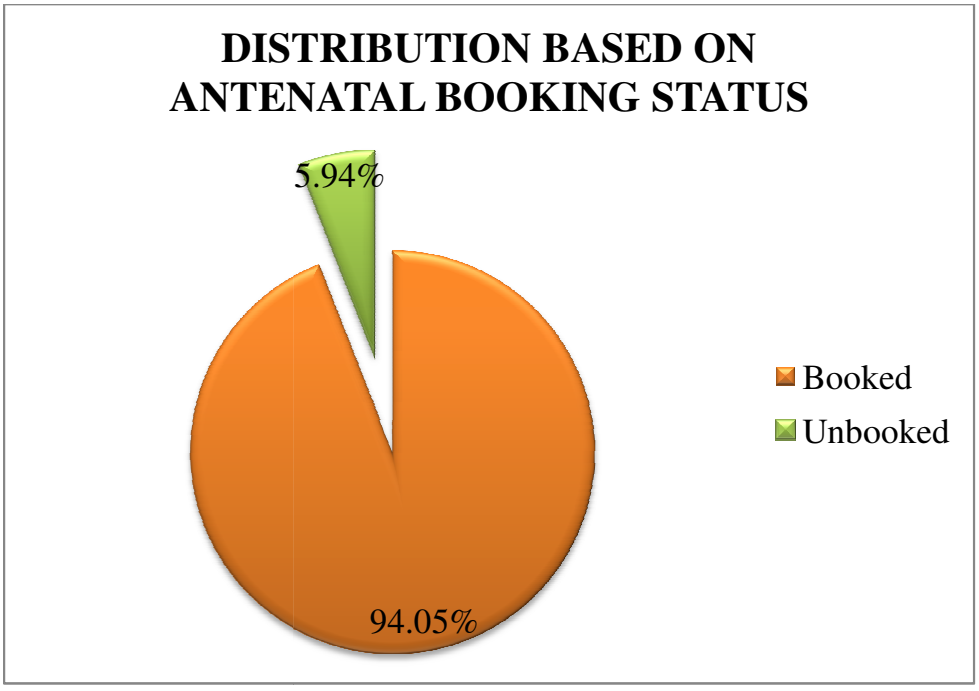


## TABLE IV

## BASED ON ANTENATAL BOOKING STATUS

| | No. of Women | % |
|---|---|---|
| Booked | 95 | 94.05% |
| Unbooked | 6 | 5.94% |

In our study the number of booked women were 94.05%



## TABLE V

## BASED ON GESTATIONAL AGE

| Gestational Age In Weeks | No. of Patients (n=101) | % |
|---|---|---|
| 28-32 weeks | 27 | 26.73% |
| 33-36 weeks | 47 | 46.53% |
| 37 Weeks & Above | 27 | 26.73% |

In our study there was a higher incidence of placental abruption in women belonging to gestational age of 33 to36 weeks.

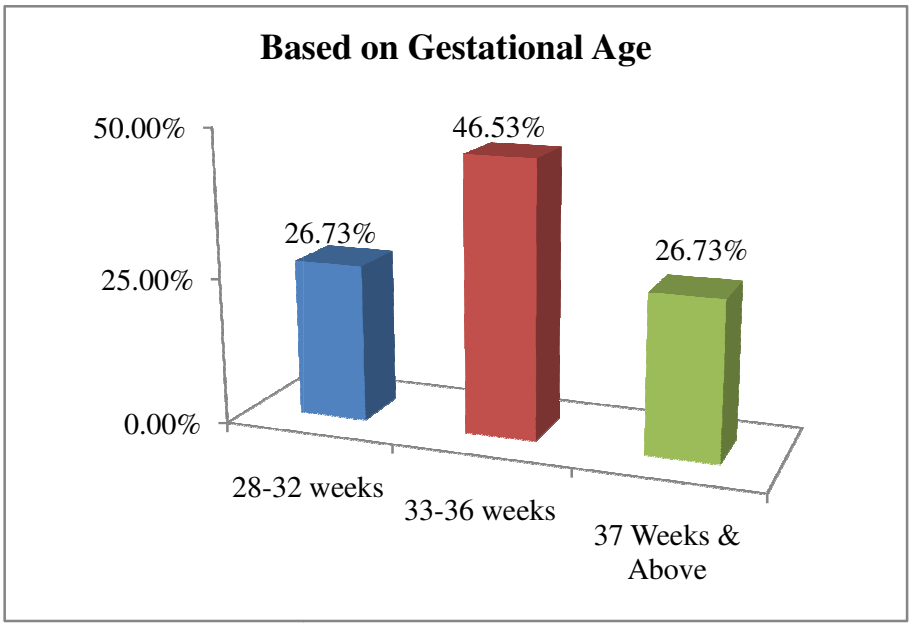


**TABLE VI**

**DISTRIBUTION BASED ON PRESENTING SIGNS AND SYMPTOMS**

| Signs and Symptoms | No. of Patients | % |
|---|---|---|
| Vaginal Bleeding | 82 | 81.18% |
| Pain Abdomen | 88 | 87.12% |
| Tense, Tender Uterus | 73 | 72.27% |
| Absent Fetal Heart | 37 | 36.63% |
| Shock | 38 | 37.62% |
| Hypertension | 62 | 61.30% |

The clinical presentation was either single or a combination of above symptoms and signs. The most common presenting sign was vaginal bleeding, followed by pain abdomen and a tense, tender uterus.

## TABLE VII

## DISTRIBUTION BASED ON RISK FACTORS

| Risk Factors | Patients withAbruption | Other patients with APH | Tests | P-value |
|---|---|---|---|---|
| Hypertensive Disorders | 62 (61.30%) | 19 | Chi-square (p<0.0000001) | significant |
| Anaemia | 76 (75.24%) | 20 | Chi-square (p<0.0000001) | significant |
| Trauma | 2 (1.98%) | 0 | Fisher Exact (p=0.5566) | Not significant |
| Hydramnios | 4 (3.96%) | 1 | Fisher Exact (p=0.4446) | Not significant |
| Short Cord | 3 (2.97%) | 1 | Fisher Exact (p=0.7103) | Not significant |
| Multiple Pregnancy | 2 (1.98%) | 2 | Fisher Exact (p>0.9999) | Not significant |
| Prev. H\o Abruption | 3 (2.97%) | - | - | - |
| Uterine Anomalies | - | - | - | - |
| Smoking and Cocaine | - | - | - | - |

Hypertensive disorders of pregnancy in the form of pregnancy induced hypertension accounted for 56 cases while 6 cases were due to chronic hypertension

.From the above table we infer that anaemia and hypertension are the two significant associated factors.

1. The patients with anaemia are having ten times risk for developing abruption,(ODDS RATIO =10.64)

2. The patients with hypertension have five times more risk of developing abruption.(ODDS RATIO=5.941)

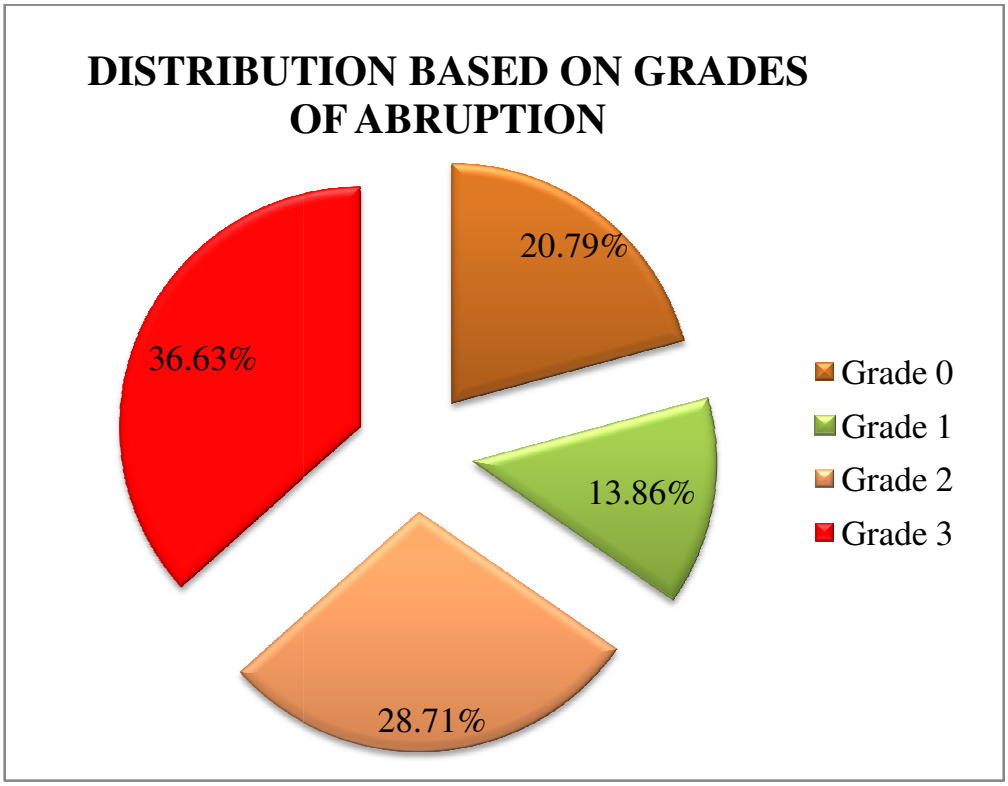
There were 2 cases of trauma abdomen which was due to a fall from a height. There were 4 cases of hydramnios. Short umblical cord was recognised postnatally in 3 cases of abruption. There were 2 cases of twin gestation in the study group. No significant anomalies could be made out in the study group. Three of the women gave a previous history of abruption of which had a short interpregnancy interval of one year. Drug abuse history was not obtained from any of the women in the study group.

## TABLE VIII

## DISTRIBUTION BASED ON GRADES OF ABRUPTION

| Grades | No. of Patients | % |
|---|---|---|
| Grade 0 | 21 | 20.79% |
| Grade 1 | 14 | 13.86% |
| Grade 2 | 29 | 28.71% |
| Grade 3 | 37 | 36.63% |

Grades of abruption were based on Page's classification. Grade 3 abruption which is a severe grade is seen in 37patients.These patients had invariably fetal demise. Some of them developed maternal complications also.

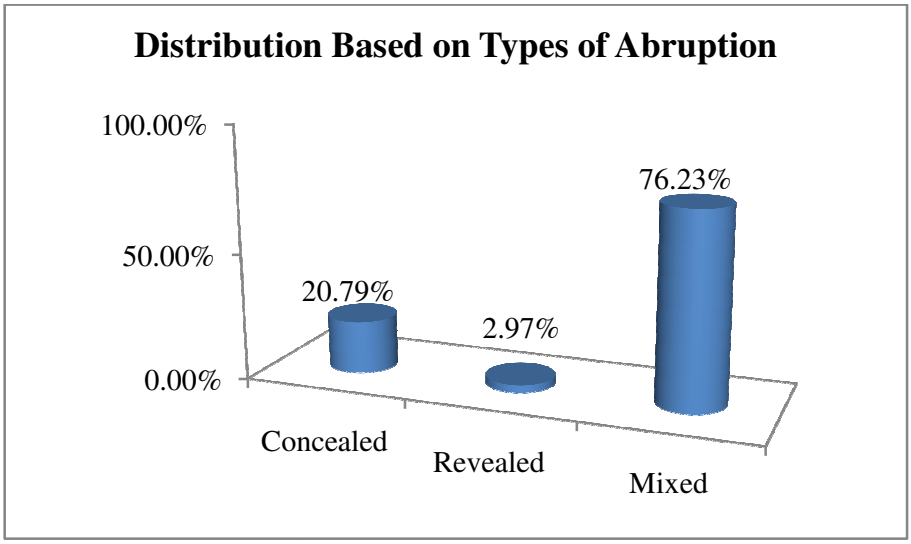


## TABLE IX

## DISTRIBUTION BASED ON TYPES OF ABRUPTION

| Distribution Based on Types of Abruption | | |
|---|---|---|
| **Types** | **No. of Patients** | **%** |
| Concealed | 21 | 20.79% |
| Revealed | 3 | 2.97% |
| Mixed | 77 | 76.23% |

The maximum number of cases were in the mixed type. Patients with onely revealed type of abruption was only 2.97%.

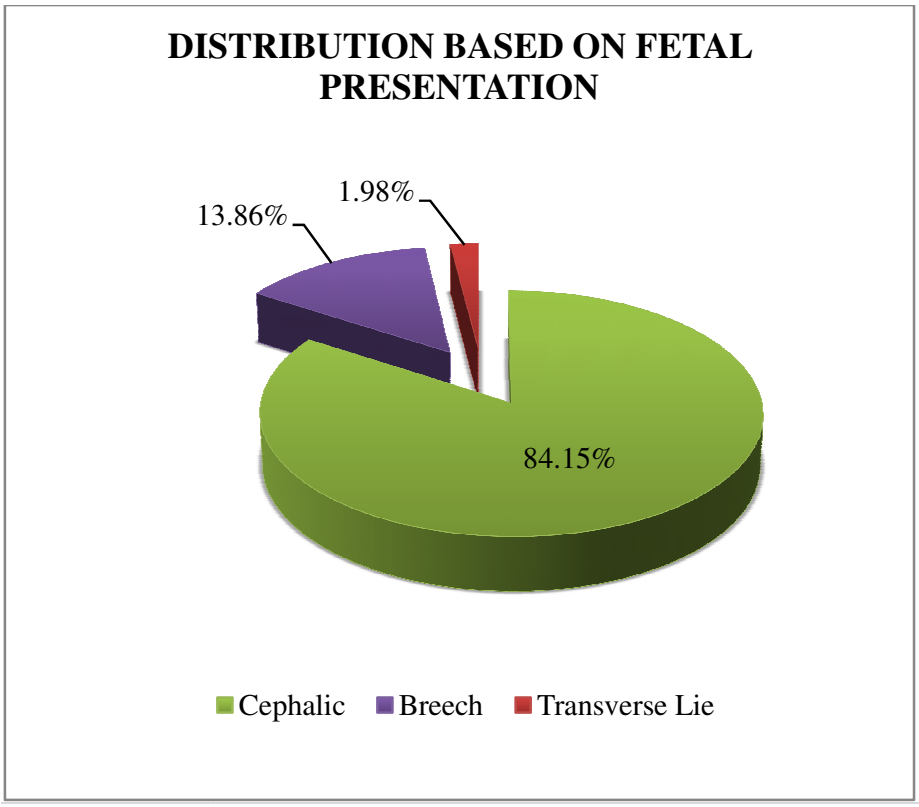


## TABLE X

## DISTRIBUTION BASED ON FETAL PRESENTATION

| Presentation | No. of Patients | % |
|---|---|---|
| Cephalic | 85 | 84.15% |
| Breech | 14 | 13.86% |
| Transverse Lie | 2 | 1.98% |

Majority of cases were in cephalic presentation. The non cephalic presentations were due to prematurity.

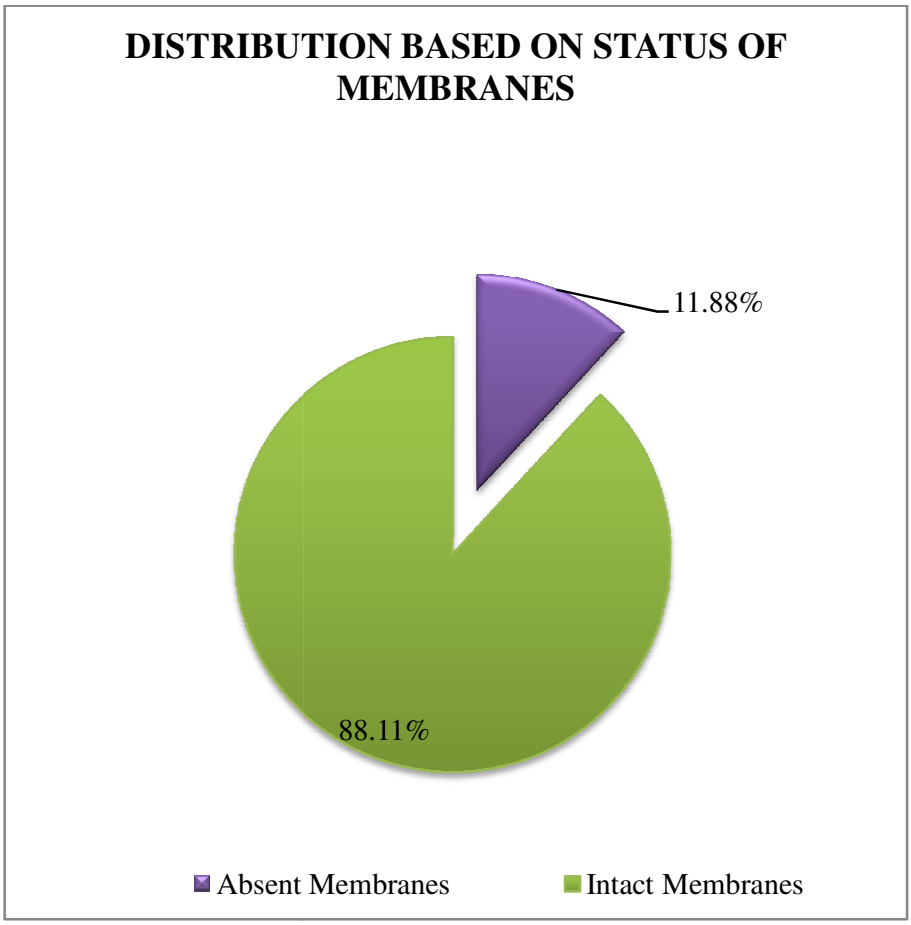


## TABLE XI

## DISTRIBUTION BASED ON MEMBRANE STATUS

| Status of Membrane on Admission | No. of Patients | % |
|---|---|---|
| Absent Membranes | 12 | 11.88% |
| Intact Membranes | 89 | 88.11% |

Most of them had intact membranes on admission

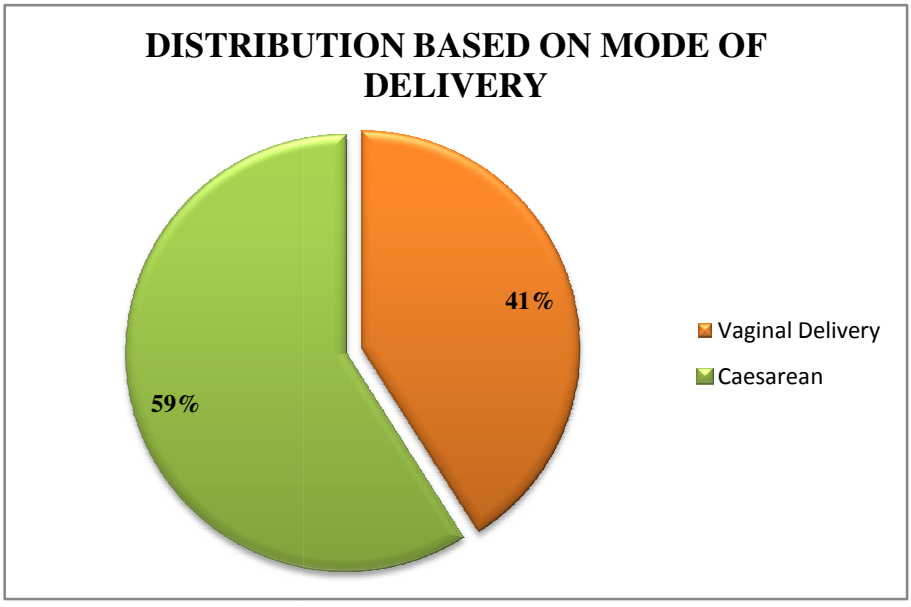


## TABLE XII

## DISTRIBUTION BASED ON MODE OF DELIVERY

| Mode of Delivery | No. of Patients (n=100) | % |
|---|---|---|
| Vaginal Delivery | 41 | 41% |
| Caesarean | 59 | 59% |

There were 100 deliveries among 101 cases. One maternal death occurred antenatally with fetus in utero. Among the 100 deliveries there were 41 vaginal deliveries and 59 caesarean deliveries. Out of the 41 vaginal deliveries Grade 0 abruption diagnosed retrospectively constituted 18 cases.



## TABLE XIII

## BREAK UP OF THE COURSE OF VAGINAL

## DELIVERIES IS SHOWN IN THE TABLE BELOW

| Nature of Progress of Vaginal Delivery | Vaginal Deliveries (n=23) | % |
|---|---|---|
| Spontaneous | Nil | Nil |
| ARM | 3 | 13.04% |
| ARM + Oxytocin | 15 | 65.21% |
| Prostaglandin Induction | 3 | 13.04% |

Among 41 vaginal deliveries excluding the18 Grade 0 cases the progress of labour in remaining 23 were studied. Three of them were delivered by amniotomy alone, fifteen of them needed an oxytocin infusion .Three of them needed an induction with misoprostol.

## TABLE XIV

## BREAKUP OF CAESAREAN SECTION EXCLUDING GRADE '0' CASES (3 CASES)

| | **No. of Patients (n=56)** | **%** |
|---|---|---|
| ARM + Oxytocin followed by LSCS | 20 | 35.71% |
| Direct LSCS | 36 | 64.28% |

Excluding the 3Grade 0 cases, out of the remining 56 cases 36 patients had direct caesarean section without attempting for a vaginal delivery. Most of them were with a history of previous LSCS or grade 2 abruptions with fetal distress.

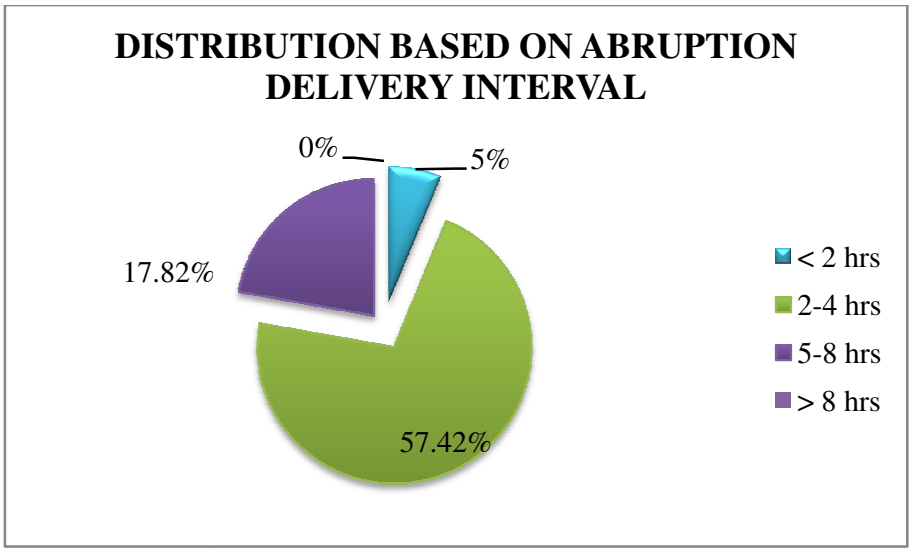
The remaining 20 patients had a Caesarean done after an attempt for vaginal delivery with amniotomy and oxytocin

## TABLE XV

## DISTRIBUTION BASED ON ABRUPTION DELIVERY INTERVAL

| Abruption Delivery Interval | No. of Women (n=80) | % |
|---|---|---|
| < 2 hrs | 5 | 6.25% |
| 2-4 hrs | 54 | 57.42% |
| 5-8 hrs | 17 | 21.25% |
| > 8 hrs | - | - |

Most of the patients were delivered with in 2-4 hours of abruption.

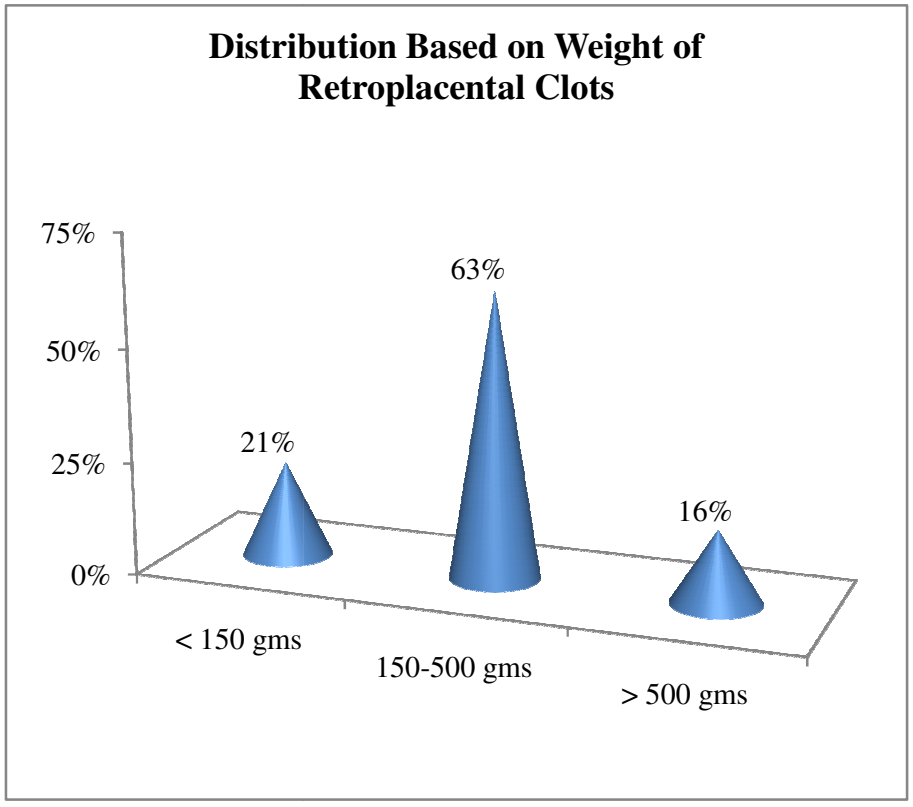


## TABLE XVI

## DISTRIBUTION BASED ON WEIGHT OF RETROPLACENTAL CLOTS

| Weight of RP Clots | Total No. of Cases (n=100) | % |
|---|---|---|
| < 150 gms | 21 | 21% |
| 150-500 gms | 63 | 63% |
| > 500 gms | 16 | 16% |

Majority of patients had retroplacental clots weighing 150-500gms.



## TABLE XVII

## DISTRIBUTION BASED ON MATERNAL COMPLICATIONS OF ABRUPTION

| Maternal Complications | Total No. of Cases (n=101) | % |
|---|---|---|
| Hypovolemic Shock | 38 | 37.62% |
| Coagulation Failure | 24 | 23.76% |
| Renal Failure | 4 | 3.96% |
| Post Partum Haemorrhage | 35 | 34.65 |
| Couvelaire Uterus | 6 | 5.94% |

Hypovolemic shock was the major complication noted. It was corrected with crystalloids, whole blood, transfusion. Coagulation failure detected by our coagulation tests was corrected by fresh frozen plasma as well as whole blood transfusions. Renal failure was corrected by correction of depleted intravascular volume and in none of them a haemodialysis was necessary.

Postpartum haemorrhage was medically managed in most of cases. Only 1 patient had a caesarean hysterectomy. In patients who had a caesarean delivery there were 6 cases of couvelaire uterus.

## TABLE XVIII

## PERINATAL MORTALITY IN VAGINAL & CAESAREAN DELIVERIES

| Mode of Delivery | Alive | Dead | Total Perinatal Mortality |
|---|---|---|---|
| Vaginal Delivery (41) | 24 | 17 | 50 |
| Caesarean (59) | 28(1T) | 33(1T) | |

Tables 19 and 20 give a break up for fetal fate based on fetal weight and route of delivery.

## TABLE XIX

## PERINATAL MORTALITY IN VAGINAL DELIVERY BASED ON BIRTH WEIGHT

| Birth Weight | <1 kg | 1000 – 1499 gms | 1500 – 2499 gms | > 2500gms | | Total Perinatal Mortality |
|---|---|---|---|---|---|---|
| Infants alive on admission | - | 1 | 7 | 17 | 25 | 15 |
| IUFD | 2 | 1 | 7 | 3 | 13 | |
| Still Birth | - | 1 | - | - | 1 | |
| Neonatal Death | - | 1 | - | - | 1 | |
| Infants Survived | - | - | 7 | 17 | 24 | |

## TABLE XX

## PERINATAL MORTALITY IN CESAREANDELIVERY

## BASED ON BIRTH WEIGHT

| Birth Weight | <1 kg | 1000 – 1499 gms | 1500 – 2499 gms | > 2500gms | | Total Perinatal Mortality |
|---|---|---|---|---|---|---|
| Infants alive on admission | - | 2 | 20(1T) | 14 | 36 | 33 |
| IUFD | 5 (1T) | 3 | 9 | 7 | - | |
| Still Birth | - | 1 | - | - | - | |
| Neonatal Death | - | 1 | 4 | 3 | - | |
| Infants Survived | - | 1 | 16 | 11 | 28 | |

Among fetuses weighing 1-1.499 kg, 3 were alive on admission and4 were dead in utero. Among the live foetuses of this group one was delivered vaginally and the same died on second day of life. The rest 2 were delivered by caesarean section and one of them died after 6 hours of life. There were 4 intrauterine fetal deaths in this group. Among them for 3 cases Caesarean delivery was done for maternal indications. There were 2 stillbirths in this group and one was delivered vaginally and the other by caesarean section

In the group weighing 1500-2499 gms 27 fetuses were alive on admission .Among them 20 fetuses were delivered by Caesarean section including a twin delivery. The 7 babies delivered vaginally survived. Out of the 20 delivered by Caesarean section16 of them survived and there were 4 neonatal deaths. There were 16 IUFDs in this group due to severe abruption. Out of them7 could be delivered vaginally.9 of them were delivered by caesarean because most of them had a previous history of caesarean.

In the group with birth weight more than 2500 grams there were 44 babies .Among them 7 delivered vaginally survived. There were 3 IUDs delivered vaginally and for 7 IUDs Caesarean section has to be done for maternal reasons. Among 14 babies delivered by Caesarean section only 11 survived.

**MATERNAL MORTALITY:**

Among the 101 cases of placental abruption, there were 2 maternal deaths. One patient was a 22 year old G5P1A3LOwith 31 weeks of gestation referred from another medical college with severe preeclampsia, bleeding p/v 2 hours duration ,placental abruption with absent fetal heart sounds with coagulation, liver, renal failure. She died within 2 hours of admission before delivery.

The other patient was a 30 year female, G2P1L1with a history of previous Caesarean section presenting at 29 weeks of gestation with bleeding P/V. She was delivered by Caesarean section a dead born female fetus of 1.6 kg after initial stabilisation of the condition. The admission delivery interval was1 hour. There was Couvelaire uterus at laparotomy but it contracted with uterotonics. There was 650 grams of retro placental clots. After surgery, there was bleeding from the surgical site and reduced renal output .Whole blood and fresh frozen plasma were transfused. Even after intensive care and expert management she died 29 hours after surgery.

# DISCUSSION OF THE RESULTS:

## TABLE XXI

## COMPARISON OF INCIDENCE IN VARIOUS INSTITUTION

| Study | Total No. of Cases | No. of Cases of Abruption | Incidence |
|---|---|---|---|
| B.N. Purandare,[33] NowrosjeeWadia Maternity Hospital Mumbai | 80,419 | 518 | 0.63% |
| VVH, Bangalore[34] | 10,467 | 202 | 1.93% |
| Present Series, IOG, Chennai | 13,239 | 101 | 0.76% |

Our study has an incidence comparable to that of the study by B.N. Purandare's study at Mumbai. Also the variation in incidence is due to the different diagnostic criteria applied for diagnoses.

## TABLE XXII

## COMPARISON OF AGE GROUP DISTRIBUTION IN VARIOUS INSTITUTION

| Age Group in Years | G.S. Mondal, Eden Hospital, Culcutta[35] | NowrosjeeWadia Maternity Hospital, Mumbai[33] | VVH, Bangalore[34] | Present Series, IOG Chennai |
|---|---|---|---|---|
| < 20 years | 8.33% | 10.52% | 23% | 6.93% |
| 21-25 years | 44.4% | 24.73% | 36% | 51.48% |
| 25-30 years | 20.7% | 37.36% | 30% | 34.65% |
| 31-35 years | 15.7% | 23.15% | 9% | 5.94% |
| > 35years | 3.72% | 4.2% | 2% | 0.99% |

When age was compared the highest incidence was in the 21-25 years age group. The reason is more number of women who delivered in our institute belong to this age group. Higher incidence was also seen in Mondol G.S. studies and VVH, Bangalore studies

## TABLE XXIII

## COMPARISON OF DISTRIBUTION OF CASES BASED ON PARITY

| Parity | NowrosjeeWadia Maternity Hospital, Mumbai[33] | VVH, Bangalore[34] | Present Series, IOG Chennai |
|---|---|---|---|
| Primi | 11.57% | 19.00% | 38.61% |
| Multi | 90.41% | 81% | 61.38% |

The incidence of abruption was more in multipara than primipara. In the other 2 studies compared there is great increase in incidence in multipara. But in our study there is not too much of difference. Anyhow parity seems to be a risk factor

## TABLE XXIV

## COMPARISON OF DISTRIBUTION BASED ON BOOKING STATUS

| Parity | G.S. Mondal, Eden Hospital, Culcutta[35] | VVH, Bangalore | Present Series, IOG Chennai |
|---|---|---|---|
| Booked | 40% | 5% | 94.05% |
| Unbooked | 60% | 95% | 5.94% |

Compared to other 2 studies where unbooked cases are a majority. In our study booked cases account for a maximum. This is due to the extensive primary health care provided by our state government.

## TABLE XXV

## COMPARISON OF DISTRIBUTION BASED ON GESTATIONAL AGE

| Studies | 28-32 weeks | 33-36 weeks | 37 weeks & Above |
|---|---|---|---|
| NowrosjeeWadia Maternity Hospital, Mumbai | 25.78% | 13.68% | 55.26% |
| VVH, Bangalore | 36% | 43% | 21% |
| IOG, Chennai | 26.73% | 46.53% | 26.53% |

Maximum incidence of abruption is seen among gestational age 33-36 weeks. This is comparable to the study at VVH, Bangalore. Most of them of the late preterm group.

## TABLE XXVI

## COMPARISON OF SIGNS & SYMPTOMS IN VARIOUS INSTITUTIONS

| Signs & Symptoms | Lakshmi Ashar, Mumbai[44] | VVH, Bangalore | IOG, Chennai |
|---|---|---|---|
| Vaginal Bleeding | 74.17% | 85% | 81.18% |
| Pain Abdomen | 54.26% | 65% | 87.12% |
| Tense, Tender Uterus | N.A. | 45% | 72.27% |
| Absent Fetal Heart | N.A. | 66% | 36.63% |
| Shock | 3.79% | 20% | 37.62% |
| Hypertension | N.A. | 27% | 61.3% |
| Anaenia | N.A. | 80% | 75.24% |

All3 studies show vaginal bleeding to be the most common presenting symptom. Hypertensive disorders seem to be the most common risk factor in all the 3 studies. Compared to the other two studies, shock was more prevalent in our study group.

## TABLE XXVII

## COMPARISON OF RISK FACTORS IN VARIOUS INSTITUTIONS

| Signs & Symptoms | Lakshmi Ashar, Mumbai | VVH, Bangalore | IOG, Chennai |
|---|---|---|---|
| Hypertensive Disorders | 40% | 20% | 61.3% |
| Trauma | 0.23% | 1% | 1.98% |
| Short Cord | Nil | 1% | 2.97% |
| Hydramnios | 0.47% | 2% | 3.96% |
| Multiple Pregnancy | Nil | 5% | 1.98% |
| Anaemia | N.A. | 80% | 75.24% |

Hypertensive disorder of pregnancy remain the most important contributory risk factor in all the three studies.

The incidence of trauma was in the lower range in all three studies.

Hydramnios associated with abruption was higher in our study.(3.96%) compared to the 2% and 0.4% seen in VVH, Bangalore studies and Lakshmi Ashar Mumbai studies.

The prevalence of anaemia also seems to be comparable to that of the VVH, Bangalore study.

## TABLE XXVIII

## COMPARISON BASED ON TYPES OF ABRUPTION

| Signs & Symptoms | Lakshmi Ashar, Mumbai[44] | VVH, Bangalore[42] | IOG, Chennai |
|---|---|---|---|
| Mixed | 66.4% | 88% | 76.23% |
| Concealed | 25.7% | 2% | 20.79% |
| Revealed | 7.9% | 10% | 2.97% |

Our study showing a higher incidence of mixed type of abruption (76.23%) is comparable to the incidence quoted by VVH, Bangalore studies (88%) and Lakshmi Ashar, Mumbai (66.4%).

In our study the number of vaginal deliveries was 41% and caesarean deliveries 59%

Various authors have reported a caesarean section rate as follows:

Hibbard B.M. and Jeffcoate[30]-8.6%

Lakshmi Ashar, Mumbai[36]-0.5%

Mudaliar A.L. and Menon-3.1%

V.V. Hospital Bangalore[34]-16%

Present IOG series-59%

Our caesarean section rate has been high in an effort to salvage the fetus. Also the maternal morbidity due to caesarean has very well decreased now a day due to the advent of higher anbtibiotics, heamotological facilities. So this increased number of Caesarean sections has definitely brought down our maternal and perinatal mortality.

## TABLE XXIX

## COMPARISON OF MATERNAL DEATHS AND PERINATAL DEATHS FROM VARIOUS STUDIES

| Studies | Maternal Deaths | Perinatal deaths |
|---|---|---|
| Lakshmi Ashar[36] | 1.6% | 87.8% |
| B.N. Purandare[33] | 0.57% | 79.5% |
| Mondal GS[35] | 6.48% | 60.91% |
| VVH, Banglore[34] | 6% | 80% |
| IOG, Chennai | 1.98% | 48% |

The perinatal mortality is very low when compared to other studies .This may be due to the improved neonatal care in our institution and the timely caesarean section done to salvage the fetuses.

The maternal mortality is 1.98% and is comparable to other institutions. This is due to delay in admissions after the occurrence of abruption as well the end organ damage due to severe grades of abruption

# SUMMARY:

- The incidence of APH was 191 among 13329 deliveries.

  ABRUPTIO PLACENTAE: 0.76%

  PLACENTA PREVIA: 0.5%

  OTHERS: 0.09%
- Abruptio placentae had the highest incidence among patients with antepartum haemorrhage.
- Highest incidence of placental abruption was seen between age group 21-25 years. The lowest age of placental abruption in our study was 18 years and the highest was 38 years.
- The majority of cases were multipara compared to primi
- Maximum incidence of abruption occurred between 33-36 weeks of gestation.
- Booked antenatal cases constituted the majority because of the increased primary health care produced by the State Government.
- The predominant presenting symptom was vaginal bleeding followed by pain abdomen, tense, tender uterus.
- Majority were of Grade 3 abruptions followed by Grade2 and Grade0.

- Most of the patients reported in 2-4 hours of onset of symptoms. This is due to the increased free transport facilities for the patient arranged by our Government.
- Coagulation abnormalities were present in 23.7% of patients where as shock was present in 37.62%of patients. Renal failure was seen in3.96 % of patients. Postpartum haemorrhage was seen in 34.65% patients and couvelaire uterus at caesarean section seen in 5.94%
- Among the vaginal deliveries 23 cases excluding Grade 0 abruption 65.21% were delivered by ARM and Oxytocin and 13.04% by prostaglandins and 13.04% with artificial rupture of membranes only.
- 41 patients had a vaginal delivery and59 patients had a caesarean delivery. There were only 8 neonatal deaths (13.55%) in babies delivered via caesarean section and one neonatal death following the vaginal deliveries.
- Overall perinatal death was 48% where in the perinatal mortality for vaginal delivery was41.46 % and the same for caesarean section was55.93%
- Among the caesarean deliveries excluding Grade 0 cases, 35.71% were done following an ARM and oxytocin

acceleration whereas 64.28 % were done directly without allowing for vaginal delivery.

- There were 2 maternal deaths in our study and the 2 of them could have been prevented if they had earlier admission.
- In Grade 1, 2 abruptions there were 0,2 neonatal deaths respectively. So the route of delivery has not influenced the fetal salvagability.
- When fetal outcome was analysed by route of delivery and birth weight, in foetuses weighing 1500-2499 gms the fetal salvage by vaginal route was7/7( 100%) and that by caesarean section was 16/20( 80%).In those foetuses weighing more than 2500 grams the salvagability by vaginal delivery was100% and that by caesarean section was11/14 78(57%).

# CONCLUSION:

Our study shows that abruptio placentae still represents a potentially serious obstetric emergency, that has an impact on fetalhealth, neonatal morbidity mortality as well as maternal health and mortality.

The major risk factors identified in our study are hypertensive disorders and anaemiain pregnancy. So management directed at identifying these risk factors at an earlier stage and correcting them will do a lot of help in reducing the incidence.

Majority of patients presented with Grade 3 abruption with a resultant intrauterine death of fetus. So early referrals from the peripheral institution would help to bring down the perinatal and maternal mortality.

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# ANNEXURES

## PROFORMA FOR EXAMINATION

Name

Age

IP no

Occupation

Address

Date of admission

Date of discharge

Socioeconomic status

Booked/Unbooked

Immunisation

**H/O Presenting complaints**

Period of amenorrhoea

LMP

EDD

Duration of pain abdomen

Duration of bleeding per vaginum

H/O perception of fetal movements

History suggestive of PIH/any medical disorders

H/O trauma

Other complaints

**Menstural History**

Age of menarche, details of menstrual cycles

**Marital History**

**Obstetric History**

Parity index

Last child birth

H/O Previous pregnancy

**Past history**

Past medical/Surgical history

**Family History**

**Personal History**

**GENERAL EXAMINATION**

Height

Weight

Nourishment

Temperature

Cyanosis

Pallor

Edema

Icterus

Lymphadenopathy

Pulse

Blood Pressure

Spine

Breast

Thyroid

CVS RS CNS

**OBSTETRIC EXAMINATION**

Fundal height

Abdominal Girth

State of uterus Tense/not Tense

Tender/not Tender

Acting/not acting

Lie,Presentation

Fetal heart sounds

**GENITAL EXAMINATION**

Vulva

**PER SPECULAM EXAMINATION**

Amount of bleeding

**PER VAGINAL EXAMINATION**

Consistency of cervix

Position

Effacement

Dilatation

Station of head

**INVESTIGATIONS**

Urine routine

Complete Haemogram

Bleeding time,Clotting time

Clot Observation test

Peripheral smear

Random blood sugar

HIV

HbsAg

VDRL

Renal function tests

ULTRASOUND

**MANAGEMENT**

Induction/Acceleration of labour

Method or drug used

Mode of delivery

Date and time of delivery

Indication for LSCS

Instrumental delivery

Duration of labour

Symptoms to delivery interval

Complications of third stage

**EXAMINATION OF PLACENTA**

Presence of infarcts/calcification

Weight of retroplacental clots

**PERINATAL OUTCOME**

Live/Stillborn/IUFD

Term/Preterm

Birth Weight

APGAR

NICU Admission: yes/no

Condition of baby at discharge

**MATERNAL OUTCOME**

Complications during labour

Condition of mother at discharge

Cause of death if any

**INSTITUTIONAL ETHICS COMMITTEE**
**MADRAS MEDICAL COLLEGE, CHENNAI –3**

EC RegNo:ECR/270/Inst./TN/2013 Telephone No : 04425305301
Fax : 044 25363970
Date:10.09.2013

**CERTIFICATE OF APPROVAL**

To
Dr.S.Sheba Mathavi,
MS OG Post Graduate,
Institute of Obstetrics and Gynaecology,
Egmore, Chennai-8.

Dear Dr.S.Sheba Mathavi,

The institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "A clinical study of incidence, risk factors, maternal, perinatal outcome in pregnancies with abruption placentae" No.04092013.

The following members of Ethics Committee were present in the meeting held on 10.09.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

# Patient Consent Form

Study Detail: A Study on "**A CLINICAL STUDY OF INCIDENCE, RISK FACTORS, MATERNAL AND PERINATAL OUTCOME IN PREGNANCIES WITH ABRUPTIO PLACENTAE"**

Study Centre: Institute of Obstetrics & Gynaecology, Egmore,
Chennai-600 008

I confirm that i have read and understood the information Sheet for the above study. I have had the opportunity to ask questions and all my questions and doubt have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that i am free to withdraw at an time, without giving any reason, without my legal rights being affected.

I understand that the Clinical study personnel, the Ethics Committee and the Regulatory Authorities will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if i withdraw from the study. I agree to this access. However, I Understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I hereby give permission to undergo completed clinical examination and diagnostic tests including haematological, biochemical, radiological tests.

I hereby consent to participate in this study.

Signature/Thumb impression:..... Place Date

of the patient

Patient's Name, Address & Ph.No:.....

Name of the Investigation:.....

Signature of the Investigator: ..... Place.....Date

Institution : .....

Signature of the Relative/Guardian.....

# ஆராய்ச்சி ஒப்புதல் படிவம்

**ஆராய்ச்சி தலைப்பு :**

கர்ப்பிணி பெண்ணிற்கு நஞ்சு கொடி பிரசவத்திற்கு முன்னரே பிரிவதால் ஏற்படும் விளைவுகள் பற்றிய ஆய்வு.

**பெயர்** : **தேதி** :

**வயது** : **உள்நோயாளி எண்** :

**பாலினம்** : **ஆராய்ச்சி சேர்க்கை எண்** :

இந்த ஆராய்ச்சியின் விவரங்களும் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியின் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆராய்ச்சியினால் ஏற்படும் நன்மைகளையும் சில பக்க விளைவுகளையும் பற்றி தெளிவாக மருத்துவர் மூலம் தெரிந்து கொண்டேன்.

நான் என் சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

..... .....

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :

இடம் :

## INFORMATION TO PARTICIPANTS

**Title** : **A clinical study of incidence, risk factors, maternal, perinatal outcome in pregnancies with abruptioplascentae.**

**Principal Investigator :** **Dr. S. SHEBA MATHAVI**

**Name of Participants :**

**Centre** : **Institute of Obstetrics & Gynaecology, Egmore.**

You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

**What is the purpose of research?**

Abruptio placentae contribute to significant maternal and fetal morbidity and mortality in our Institute. The purpose of the study is to find out the incidence of abruptio placentae, risk factors and the impact of abruption on maternal and perinatal morbidity and mortality.

**The Study Design:**

Pregnant women with gestational age more than 28 weeks admitted with complaints of bleeding per vaginum and those patients with retroplacental clots were included in the study. They are followed up both intrapartum & postpartum.

**Study Procedures:**

Pregnant women with gestational age of more than 28 weeks and those patients with retroplacental clots are categorized by their grades of abruptio placentae, Ultrasonogram and Blood investigations will be done for you. You are followed up during delivery and postpartum. The outcome will be analysed. You may have to come to the hospital for follow up.

**Possible risks to you :** Nil

**Possible benefits to you :** You will be under regular supervision and follow up.

**Possible benefits to other people:**

The results of the research may provide benefits to the society in terms of advancement of medical knowledge and / or therapeutic benefit to future patients.

**Confidentiality of the information obtained from you**

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, sponsors, Institutional Ethics Committee to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

**How will your decision to not participate in the study affect you?**

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not lose any benefits to which you are entitled.

**Can you decide to stop participating in the study once you start?**

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to discontinuation.

Signature of Investigator

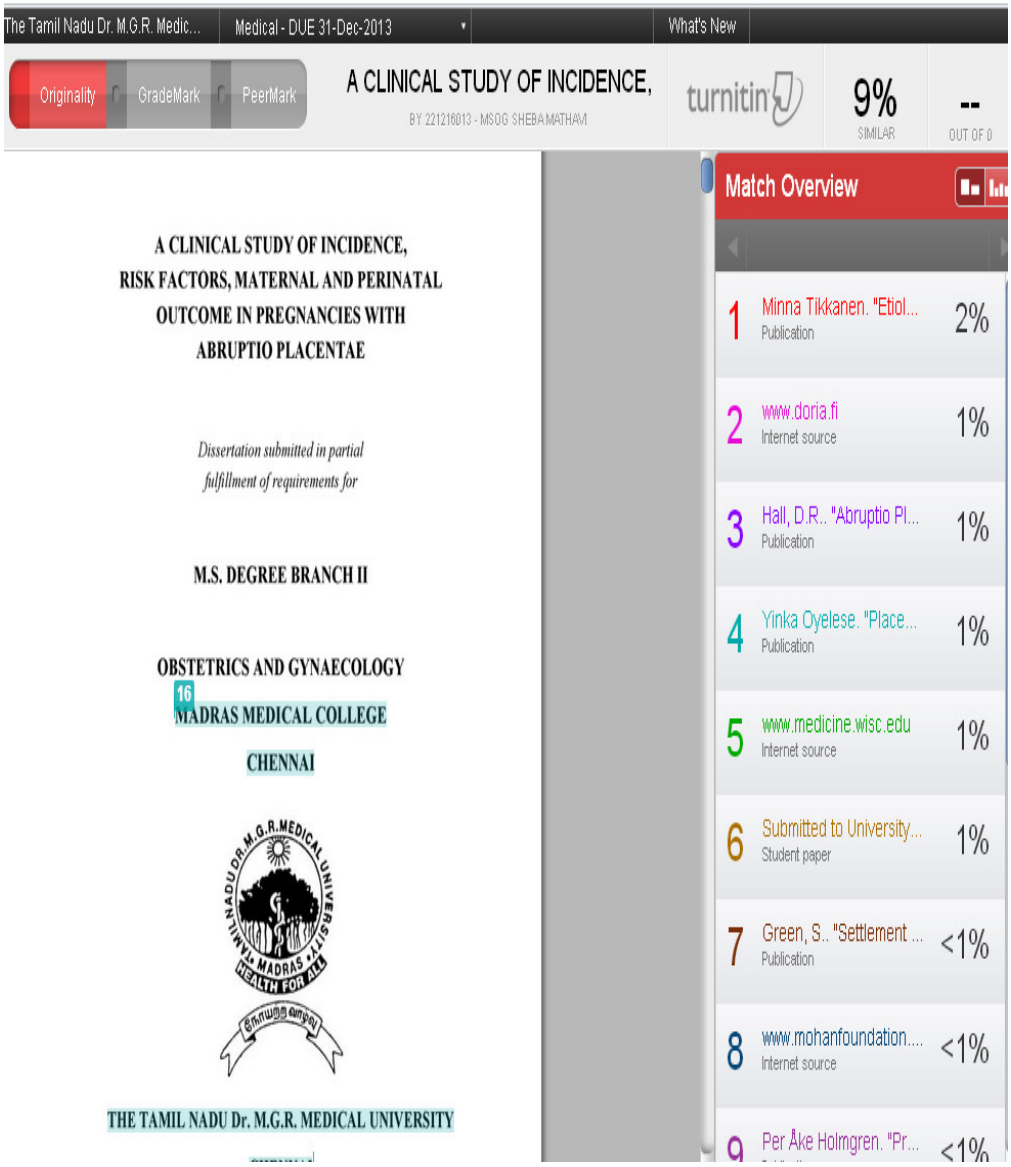
date

Signature of Participant

date

## ABBREVIATIONS

| | |
|---|---|
| LSCS | Lower segment Caesarean section |
| ARM | Artificial rupture of membranes |
| IUFD | Intrauterinefetal death |
| DIC | Disseminated Intravascular Coagulation |
| Wt | Weight |
| VVH | Vani vilas Hospital |
| APH | Antepartum Haemorrhage |
| Gest | Gestation |
| Wks | Weeks |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# MASTER CHART

| S.No. | Name | Age | IP NO | Ob. Code | B\UB | G. Age (Wks) | Maternal S/S | FH | RISK FACT | Grade | Type | Presentation | MAT. Compli. | MOD | Ab-Del. Int. | Wt. of RP Clots | Apgar Score | Baby wt. | P.O | M.M |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | RAJESWARI | 26 | 28637 | primi | B | 38 | VB+P+T | + | HT:A | 1 | M | ceph | _ | Cs | 4 | 150gms | 7?10,8/10 | 3.02 | | |
| 2 | RATHIKA | 23 | 29321 | G2P1 L1 | B | 35 | VB+P+T | - | HT,A | 3 | M | ceph | ,S,CA,PPH | Cs | 6 | 550gms | 3/10,5/10 | 2.4 | IUD | |
| 3 | SARITHA | 22 | 29321 | G2P1 L1 | B | 37 | VB+P+T | - | HT+A | 3 | M | ceph | | CS | 7 | 250gms | 2/10,5/10 | 2.06 | IUD | |
| 4 | LATHA | 20 | 28669 | PRIMI | B | 36 | VB | + | A | | | ceph | | Cs | | | 5/10,8/10 | 2.12 | | |
| 5 | VENNILA | 22 | 28797 | primi | B | 36 | VB | + | HY | | | TR.LIE | | VAG | | | 7/10,8/10 | 2.5 | | |
| 6 | MAHESWARI | 21 | 28301 | primi | B | 37 | VB | + | | | | ceph | | Cs | | | 6/10,8/10 | 2.45 | | |
| 7 | DURGA | 29 | 28672 | primi | B | 38 | VB | + | A | | | ceph | | Cs | | | 6/10,8/10 | 3.7 | | |
| 8 | VENNILA | 27 | 30046 | G3P1 LIA1 | B | 36 | VB | + | HT+A | 2 | R | ceph | | Cs | 2 | 500gms | 2/10,3/10 | 2 | ND | |
| 9 | RAJESWARI | 23 | 30126 | G3P1 L1A1 | B | 35 | VB+P+T | - | HT+A | 3 | M | ceph | S,CA,PPH | VAG | 6 | 550gms | 0/10 | 2 | IUD | |
| 10 | HEMALATHA | 24 | 29707 | G3P1 LIA1 | B | 36 | VB | + | | | | OBLIQUE | S | Cs | | | 4/10,7/10 | 3 | | |
| 11 | SUBHA | 26 | 29711 | primi | B | 31 | VB | | A | | | ceph | | Cs | | | 7/10,8/10 | 2.7 | | |
| 12 | THANGAM | 27 | 29389 | G2P1 L1 | B | 38 | VB | + | | | | ceph | | Cs | | | 6/10,8/10 | 1.9 | ND | |
| 13 | KAVITHA | 21 | 29410 | primi | B | 35 | VB | + | | | | ceph | | Cs | | | 5/10,7/10 | 2.6 | | |
| 14 | VALLI | 32 | 29680 | primi | B | 36 | VB | + | A | | | ceph | | VAG | | | 6/10,8/10 | 2.2 | | |
| 15 | MEERA | 21 | 29664 | G2P1 L1 | B | 37 | VB | + | | | | ceph | | Cs | | | 7/10,8/10 | 2.4 | | |
| 16 | BANU | 26 | 29680 | G2A1 | B | 37 | P | - | | 0 | C | ceph | | VAG | | 450gms | 0/10 | 3 | IUD | |
| 17 | RAMYA | 25 | 29681 | primi | | 32 | VB+P | + | | | | ceph | | VAG | | | | 3 | | |
| 18 | DHANALAKSMI | 27 | 29797 | G2P1 L1 | B | 32 | VB | + | | | | TR.LIE | | Cs | | | 6/10,7/10 | 1.76 | | |
| 19 | SUBASHINI | 26 | 31164 | G4P2 L2A1 | B | 33 | VB+P+T | _ | HT+A+RE | 0 | M | ceph | S,PPH | CS | 4.5 | 100gms | 5/10,7/10 | 1.8 | | |
| 20 | PALAIYATHAL | 24 | 31494 | G2P1 L0 | B | 36 | VB+P+T | _ | HT+A | 3 | M | ceph | S,PPH | CS | 1 | 550gms | 0/10 | 2.8 | IUD | |
| 21 | ANNE SUJI | 24 | 29667 | primi | B | 21 | | + | | | | ceph | | | | | | 2.4 | | |

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| 22 | MUTHUMATHY | 27 | 31503 | primi | B | | | -I | | | | ceph | | | | | | 2.8 | | |
| 23 | RATHINAM | 25 | 31511 | primi | B | 35 | VB | + | SC | | | ceph | S | VAG | | | | 2.25 | IUD | |
| 24 | VIDYA | 26 | 31524 | G2P1L1 | B | 32 | VB | + | A | | | ceph | | Cs | | | | | | |
| 25 | MATHURI | 28 | 31556 | G2A1 | B | 34 | VB | + | A | | | ceph | | VAG | | | | | | |
| 26 | Sumathy | 25 | 31605 | G2P1L1 | B | 38 | VB | _ | | 3 | M | ceph | S,CA,PPH | VAG | 2 | 400gms | 0/10 | 2.25 | IUD | |
| 27 | SATHYA | 22 | 31611 | G2A1 | B | 36 | VB+P+T | + | | 1 | M | ceph | | Cs | 3 | 100gms | 7/10,8/10 | 2.25 | | |
| 28 | JEYAKODI | 30 | 31540 | G2P1L1 | B | 36 | VB | + | A | | | ceph | | Cs | | | | | | |
| 29 | MALARKODI | 32 | 31740 | primi | B | 37 | VB | + | A | | | ceph | | Cs | | | | | | |
| 30 | MALAR | 34 | 31628 | G2P1L1 | B | 37 | VB | + | A | | | ceph | | Cs | | | | | | |
| 31 | REVATHY | 25 | 31657 | primi | UB | 29 | VB | + | | | | ceph | | Cs | | | | | | |
| 32 | ANUCHITRA | 21 | 35487 | PRIMI | UB | 29 | VB+P+T | - | HT+A | 3 | M | ceph | S,CA,PPH | VAG | 1 | 550gms | 0/10 | 950 | IUD | |
| 33 | LAKSHMI | 28 | 35003 | G1P1L1 | B | 30 | VB+P+T | _ | HT+TW+A | 3 | M | BR,CEPH | S,PPH | Cs | 3 | 600gms | 0/10 | 500,500 | BOTHIUD | |
| 34 | ANITA | 27 | 36576 | G2P1L1 | B | 28 | VB+P+T | - | HT+A | 3 | M | ceph | ,S,CA,PPH | VAG | 2 | 500gms | 0/10 | 2.25 | IUD | |
| 35 | RADHIKA | 27 | 31706 | primi | B | 34 | VB | + | A | | | ceph | | Cs | | | | | | |
| 36 | ANSARI | 25 | 31721 | G2A1 | B | 36 | VB | + | A | | | ceph | | Cs | | | | | | |
| 37 | RAMANI | 24 | 31734 | primi | B | 38 | VB | + | HT | | | ceph | | Cs | | | | | | |
| 38 | SARANYA | 26 | 35741 | primi | B | 36 | VB | + | | | | ceph | | VAG | | | | | | |
| 39 | AMALA | 28 | 35756 | primi | B | 35 | VB | + | | | | ceph | | Cs | | | | | | |
| 40 | MENAGA | 23 | 37946 | G2P1L1 | B | 37 | | + | A | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 3 | | |
| 41 | BHUVANA | 24 | 37936 | primi | B | 39 | VB+T | + | A | 2 | M | BR | S | Cs | 4 | 200gms | 5/10,7/10 | 3.1 | | |
| 42 | DIVYA | 29 | 37950 | G2P1L1 | B | 37 | VB | + | | | | ceph | | Cs | | | | | | |
| 43 | RADHA | 32 | 37978 | G3P2L2 | B | 35 | VB | + | | | | ceph | | Cs | | | | | | |

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| 44 | PANJALI | 26 | 37952 | primi | B | 38 | | + | A | 0 | C | ceph | | VAG | | 100gms | 7/10,8/10 | 3.2 | | |
| 45 | SEETHA | 24 | 37964 | primi | B | 36 | | + | A | 0 | C | ceph | | VAG | | 400gms | 0/10 | 2.4 | | |
| 46 | SUSEELA29 | 25 | 37968 | primi | B | 37 | VB | _ | | | | ceph | | Cs | | | | | IUD | |
| 47 | SARADHA | 26 | 37971 | primi | B | 34 | VB | + | | | | ceph | | Cs | | | | | | |
| 48 | GOMATHY | 25 | 37864 | primi | B | 33 | VB | + | | | | ceph | | Cs | | | | | | |
| 49 | ARULMOZHI | 22 | 33718 | G2P1L1 | B | 39 | VB+P+T | + | HT+A+RE | 2 | M | ceph | S,PPH | Cs | 3 | 200gms | 4/10,6/10 | 3.5 | | |
| 50 | RAJESWARI | 32 | 36806 | G2P1L1 | B | 33 | VB | + | | | | BR | | Cs | | | | | | |
| 51 | DEEPA | 20 | 36765 | G2P1L1 | B | 38 | VB+P+T | + | HT+A | 2 | M | ceph | | Cs | 3 | 200gms | 5/10,7/10 | 2.4 | AD | |
| 52 | MARY | 26 | 31714 | PRIMI | B | 34 | VB | + | | | | ceph | | CS | | | | | | |
| 53 | PATTU | 25 | 31721 | G2P1L1 | B | 35 | VB | + | | | | ceph | | VAG | | | | | | |
| 54 | MALATHY | 26 | 31728 | G2P1L1 | B | 36 | P+T | + | | | | ceph | | CS | | 100gms | 6/10,7/10 | 2.8 | | |
| 55 | VASANTHA | 25 | 31740 | G2P1L1 | B | 37 | P | + | | | | ceph | | VAG | | 50gms | 7/10,8/10 | 2.5 | | |
| 56 | DURGA | 25 | 31748 | PRIMI | B | 37 | VB+P | + | | | | ceph | | CS | | | | | | |
| 57 | ELAKKIYA | 26 | 31884 | G2P1L1 | B | 34 | VB+P+T | + | HT,A | 2 | M | ceph | | CS | 2.5 | 300gms | 5/10,6/10 | 1.7 | ND | |
| 58 | MEENATCHI | 21 | 31891 | G2P1L1 | B | 28 | VB+P+T | + | HT,A | 2 | M | BR | | VAG | 3.5 | 350gms | 4/10,6/10 | 1.2 | ND | |
| 59 | GEETHA | 25 | 31839 | G2P1L1 | B | 38 | VB | + | | | | ceph | | CS | | | | | | |
| 60 | SUMATHY | 23 | 32172 | G2P1L1 | B | 38 | VB+P | + | | 0 | M | ceph | S | VAG | 3 | 100gms | 6/10,7/10 | 2.9 | | |
| 61 | VIJAYA | 26 | 2461 | PRIMI | B | 30 | VB | _ | | | | ceph | | Cs | | | | | | |
| 62 | SASIREKA | 28 | 2881 | G2P1L1 | B | 29 | VB+P+A | _ | A | 3 | M | ceph | S | VAG | 2 | 550gms | 0/10 | 1 | IUD | |
| 63 | BAGIYA | 20 | 3256 | PRIMI | B | 36 | VB | | | | | ceph | | Cs | | | | | | |
| 64 | VANI | 21 | 3213 | primi | UB | 32 | VB+P+T | _ | HT+A | 3 | M | ceph | ,,S,CA,PPH | VAG | 1.5 | 400gms | 0/10 | 1.85 | IUD | |
| 65 | VIDYA | 24 | 3310 | primi | B | 36 | P | + | | 0 | C | ceph | | VAG | | 50gms | 7/10,8/10 | 2.7 | | |
| 66 | RENGA | 29 | 3318 | primi | B | 34 | P | + | | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 1.8 | | |
| 67 | BHARATI | 24 | 3325 | primi | B | 34 | VB+P | + | | | | ceph | | | | | | | | |
| 68 | NISHA | 23 | 3327 | primi | B | 37 | VB+P | + | TW | | | ceph | | | | | | | | |

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| 69 | BHARATI | 28 | 3228 | G3P2L2 | B | 35 | VB+P+T | - | HT+A+SC | 3 | M | ceph | S,CA,PPH | CS-CO+HYS | 3.5 | 550gms | 0/10 | 1.5 | IUD | |
| 70 | JULIE | 28 | 3332 | G2P1L1 | B | 35 | VB+P+T | _ | HT+HY | 3 | M | BR | S,CA,PPH | Cs | 4 | 400gms | 0/10 | 1.8 | IUD | |
| 71 | LAKSHMIDEVI | 30 | 3479 | primi | B | 33 | VB+P+T | - | | 3 | M | BR | S,CA,PPH | CS | 5 | 450gms | 0/10 | 2.8 | IUD | |
| 72 | JYOTY | 30 | 3586 | G2P1L1 | B | 29 | VB+P+T | - | HT+A | 3 | M | ceph | S,CA,RF,PPH | CS | 6 | 500gms | O/10 | 750 | IUD | DIED |
| 73 | SAHAYA | 24 | 3751 | primi | B | 34 | VB+P+T | + | | 1 | R | ceph | | CS | 4 | | 5/10,7/10 | 2.5 | AD | |
| 74 | MANJU | 26 | 3820 | G2P1L1 | B | 35 | VB | + | | | | ceph | | CS | | | | | | |
| 75 | LAKSMI | 22 | 3578 | primi | B | 36 | VB | + | | | | ceph | | CS | | | | | | |
| 76 | KALAIYARASI | 26 | 4293 | G2P1L1 | B | 33 | VB+P+T | + | HT+TR | 2 | M | ceph | | CS | 4 | 300gms | 0/10 | 1.2 | SB | |
| 77 | DHANALAKSMI | 32 | 4435 | G7P1L1A5 | UB | 28 | VB+P+T | _ | HT+A | 3 | M | ceph | S,PPH | CS | 5 | 450gms | 0/10 | 1 | IUD | |
| 78 | RAMYA | 27 | 4756 | G3A2 | B | 32 | VB+P+T | + | | 2 | M | BR | | CS | | 350gms | 4/10,7/10 | 1.6 | AD | |
| 79 | MANJULA | 24 | 4760 | G3P1L1A1 | B | 35 | P | + | | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 1.62 | AD | |
| 80 | GOMATHY | 23 | 4764 | G2P1L1 | B | 37 | P | + | | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 3 | | |
| 81 | MADHURI | 26 | 4781 | primi | B | 36 | VB | + | | | | ceph | | | | | | | | |
| 82 | AMALA | 26 | 4787 | primi | B | 37 | VB+P | + | | 3 | M | ceph | | CS | | | 6/10,8/10 | 2.7 | IUD | |
| 83 | DHANALAKSMI | 25 | 4857 | G2A1 | B | 36 | VB+P+t | + | HT+A | 3 | M | ceph | | CS | 3 | 250gms | 5/10,7/10 | 2.3 | | |
| 84 | JEEVARANI | 22 | 5738 | G3P1L1A1 | B | 30 | VB+P+T | _ | HT+A | 3 | M | BR | S | CS-CO | 4 | 450gms | 0/10 | 1.6 | IUD | |
| 85 | SUGUNA | 25 | 5918 | G2P1L1 | B | 39 | VB+P+T | _ | HT | 3 | M | ceph | S,CA,RF,PPH | CS | 6 | 550gms | 0/10 | 3.2 | IUD | |
| 86 | GAYATHRI | 21 | 5932 | primi | B | 36 | VB+P+T | _ | HT | | | BR | S,CA,PPH | | 5 | 550gms | 0/10 | 1.9 | IUD | |
| 87 | SHANTI | 26 | 5922 | G2A1 | B | 35 | VB | + | A | | | ceph | | CS | | | | | | |
| 88 | CHITRA | 31 | 5974 | G2P1L1 | B | 36 | VB | + | A | | | BR | | CS | | | | | | |
| 89 | MALAR | 26 | 6495 | primi | B | 37 | VB+P=T | _ | HT,A | 3 | M | ceph | S,CA,PPH | CS | 4 | 400gms | 0/10 | 1.5 | IUD | |
| 90 | AMUL | 26 | 6924 | primi | B | 37 | VB+P+t | + | HT | 2 | M | ceph | | CS | 4 | 200gms | 5/10,7/10 | 3.5 | | |
| 91 | DEVI | 23 | 7136 | G2P1L1 | B | 35 | VB+P+T | + | HT+HY | 1 | M | ceph | | CS | 3 | 100gms | 6/10'8/10 | 3.3 | | |
| 92 | GIRIJA | 21 | 7905 | G2P1L1 | B | 36 | VB+P+T | + | HT+A+SC | 2 | M | ceph | | CS | 4 | 200gms | 4/10,6/10 | 2.6 | ND | |

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| 93 | RAJAM | 34 | 7932 | G2P1L1 | B | 35 | VB | + | | | | ceph | | CS | | | | | | |
| 94 | THILAKA | 32 | 7831 | primi | B | 34 | VB | + | HT | | | ceph | | CS | | | | | | |
| 95 | PARVATHY | 28 | 7965 | G2A1 | B | 35 | VB | + | | | | ceph | | CS | | | | | | |
| 96 | DEVI | 30 | 7894 | primi | B | 36 | VB | + | A | | | BR | | CS | | | | | | |
| 97 | AZEEMA | 24 | 8131 | G2P1L0 | B | 35 | VB+P+T | _ | HT+A | 3 | M | ceph | S | CS | 6 | 350gms | 0/10 | 2.7 | IUD | |
| 98 | SARANYA | 25 | 8472 | G2P1L1 | B | 31 | VB+P+t | _ | HT+A | 3 | M | BR | S | CS | 7 | 600gms | 0/10 | 1.4 | IUD | |
| 99 | PRIYA | 24 | 8476 | G3P1L1A1 | B | 36 | P | + | | 0 | C | ceph | PPH | VAG | | 100gms | 7/10,8/10 | 2.6 | | |
| 100 | REMA | 28 | 8480 | G3P2L2 | B | 38 | P | + | A | 0 | C | ceph | | VAG | | 50gms | 6/10,7/10 | 2.8 | | |
| 101 | SAROJA | 27 | 8489 | primi | B | 37 | P | + | | | | ceph | | VAG | | | | | | |
| 102 | SASIREKA | 30 | 8479 | G2P1L1 | B | 29 | VB+P+T | _ | HT,A | 3 | M | BR | S,CA,RF,PPH | CS | 5.5 | 650gms | 0/10 | 1 | IUD | |
| 103 | SIVAKAMI | 28 | 9361 | G2P1L0 | B | 35 | VB | + | HT | | | BR | | CS | | | | | | |
| 104 | KANAGA | 26 | 9462 | primi | B | 34 | VB | + | | | | ceph | | CS | | | | | | |
| 105 | VIJALAKSMI | 27 | 9465 | G2P1L0 | B | 36 | VB+P=T | - | HT,A | 2 | M | ceph | | CS | 3 | 300gms | 5/10,7/10 | 1.9 | | |
| 106 | BALASUNDARI | 29 | 9596 | G2P1L1 | B | 35 | VB+P+T | _ | HT,A | 1 | M | ceph | | CS | 2.5 | 150gms | 4/10,6/10 | 2.5 | ND | |
| 107 | MALINI | 24 | 9261 | G3P1L1A1 | B | 32 | VB+P+T | + | HT+A+SC | 2 | M | TR LIE | | CS | 3 | 200gms | 5/10,7/10 | 1.5 | | |
| 108 | DEVI | 22 | 10274 | G2P1L1 | B | 37 | VB+P+T | - | HT,A | 3 | M | ceph | S,CA,PPH | CS-CO | 4 | 300gms | 0/10 | 2.75 | IUD | |
| 109 | JEYANTI | 34 | 10962 | G2P1L1 | B | 35 | VB+P+T | - | HT | 3 | M | ceph | S,CA,PPH | CS | 4.5 | 450gms | 0/10 | 1.95 | IUD | |
| 110 | GURUSELVI | 25 | 11033 | G2P1L1 | B | 34 | VB+P+T | + | HT,TW | 1 | M | BOTHCEPH | | CS | 4 | 250gms | 6/10,7/10 | 2.3,2.2 | | |
| 111 | PARIMALA | 23 | 11964 | G3P1L1A1 | B | 32 | VB+P+T | - | HT,HY | 3 | M | BR | S,CA,PPH | CS | 6 | 550gms | 0/10 | 2 | IUD | |
| 112 | VIJAYALAKSMI | 27 | 12011 | G5P3L1A2 | B | 32 | VB+P+T | + | HT,A | 2 | M | BR | | CS | 3.5 | 300gms | 6/10,7/10 | 1.5 | | |
| 113 | MANGALAKSMI | 25 | 12887 | G3P2L2 | B | 37 | VB | + | HT | | | ceph | S,PPH | CS | | | | | | |
| 114 | NITYA | 26 | 12889 | G2P1L1 | B | 36 | P | + | A | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 2.8 | | |
| 115 | PALANIAMMAL | 28 | 12891 | primi | B | 37 | P | + | A | 0 | C | ceph | | VAG | | 50gms | 6/10,8/10 | 2.9 | | |

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| 116 | RADHIKA | 23 | 12911 | primi | B | 37 | P | + | TW | | | ceph | | VAG | | | | | | |
| 117 | SUSIMA | 24 | 12278 | primi | B | 38 | VB | + | A | | | ceph | S,PPH | CS | | | | | | |
| 118 | GOMATHY | 28 | 11766 | G3A2 | B | 33 | VB | + | HT | | | ceph | | CS | | | | | | |
| 119 | ROSY | 27 | 13850 | primi | B | 37 | VB+P+T | + | HT,A | 1 | M | ceph | | CS | 3.5 | 200gms | 6/10,8/10 | 2.8 | | |
| 120 | PUNITHA | 21 | 15208 | G2P1L1 | B | 35 | VB+T | + | HT,A | 2 | M | ceph | | CS | 2 | 350gms | 6/10,7/10 | 1.89 | | |
| 121 | BHARATI | 23 | 16717 | primi | B | 30 | VB+T | - | HT,A | 3 | M | BR | S,CA,PPH | CS | 3.5 | 400gms | 0/10 | 800 | IUD | |
| 122 | ABIRAMI | 22 | 16442 | primi | B | 38 | VB | + | | 1 | M | ceph | | CS | 3 | 200gms | 5/10,7/10 | 2.6 | | |
| 123 | PREETHI | 18 | 16662 | primi | UB | 31 | VB+P+T | + | A | 2 | M | ceph | | VAG | 2.5 | 300gms | 5/10,7/10 | 1.3 | AD | |
| 124 | SUNITA | 30 | 16668 | primi | B | 38 | VB+P+T | - | HT,A | 3 | M | ceph | S,PPH | VAG | 2 | 350gms | 0/10 | 2.8 | IUD | |
| 125 | SELVI | 21 | 17951 | primi | B | 35 | VB+P+T | + | HT,HY | 2 | M | ceph | | VAG | 1.5 | 250gms | 6/10,8/10 | 2.6 | AD | |
| 126 | DEEPA | 24 | 18170 | G2P1L1 | B | 33 | VB+P+T | - | HT,A | 3 | M | ceph | S,CA,PPH | VAG | 1 | 450gms | 0/10 | 1.7 | IUD | |
| 127 | VIDYA | 27 | 18180 | G2P1L1 | B | 37 | P | + | | 0 | C | ceph | | VAG | | 100gms | 7/10,8/10 | 2.7 | | |
| 128 | RANI | 28 | 18182 | G2P1L1 | B | 38 | P | + | | 0 | C | ceph | | VAG | | 50gms | 7/10,8/10 | 3 | | |
| 129 | BRINDHA | 29 | 18179 | primi | B | 37 | P | + | HT | | | ceph | | VAG | | | | | | |
| 130 | NADIYA | 26 | 18251 | G2P1L1 | B | 38 | VB+P | + | A | | | ceph | | CS | | | | | | |
| 131 | SATYA | 22 | 18224 | G2A1 | B | 35 | VB+P+T | + | HT,A | 2 | M | ceph | | CS-CO | 3 | 350gms | 5/10,7/10 | 2.25 | | |
| 132 | HEMALATHA | 24 | 17264 | G3A2 | B | 34 | VB | + | | | | TR LIE | | CS | | | | | | |
| 133 | ELIZABETH | 22 | 18557 | G2P1L1 | B | 29 | VB+P+T | + | HT,A | 1 | M | ceph | | VAG | 2 | | 6/10,8/10 | 2.1 | | |
| 134 | KOKILA | 23 | 18743 | G2P1L1 | B | 31 | VB | + | HT | | | ceph | | VAG | | | | | | |
| 135 | KOMALA | 23 | 18444 | G2P1L1 | B | 29 | VB | + | HT | | | ceph | | CS | | | | | | |
| 136 | DEEPA | 29 | 19266 | G3P1L1A1 | B | 35 | VB | + | | | | ceph | | VAG | | | | | | |
| 137 | SHARMILA | 22 | 18775 | G5P1L0A3 | B | 31 | VB | _ | HT,A | 3 | M | BR | HELLP,S,CA,RF | | | | | | IUD | DIED |
| 138 | VASANTHI | 23 | 19780 | G2P1L1 | UB | 29 | VB | - | HT,A | 3 | M | ceph | S,CA,PPH | CS | 3.5 | 250gms | 0/10 | 2.75 | IUD | |
| 139 | PUNITHA | 28 | 20634 | G2P1L1 | B | 37 | VB+P+T | - | HT,A | 3 | M | ceph | S,CA,PPH | CS | 5 | 350gms | 0/10 | 1.5 | IUD | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 140 | MARIAMMAL | 23 | 21173 | G2A1 | B | 31 | VB+P+T | + | HT,A | 3 | M | ceph | | CS | | 100gms | 5/10,7/10 | 2.8 | | |
| 141 | ANSARI | 26 | 21176 | G3P2L2 | B | 36 | P | + | A | 0 | C | ceph | | VAG | | 100gms | 6/10,8/10 | 3 | | |
| 142 | KALYANI | 22 | 21182 | G2P1L1 | B | 38 | P | + | A | 0 | C | ceph | | VAG | | | | | | |
| 143 | SANKARI | 26 | 21190 | primi | B | 37 | P | + | HT | | | ceph | | VAG | | | | | | |
| 144 | VINODHINI | 24 | 21187 | primi | B | 36 | P | + | HT | | | ceph | | CS | | | | | | |
| 145 | THANGAM | 20 | 21189 | primi | B | 37 | VB+P | + | HT | | | ceph | | CS | | | | | | |
| 146 | PATTU | 27 | 21195 | G2P1L1 | B | 35 | VB+P | + | | | | ceph | | CS | | | | | | |
| 147 | AGALYA | 21 | 21659 | primi | B | 32 | VB+P+T | + | HT,A | 2 | M | ceph | S,PPH | CS | 7 | 250gms | 3/10,5/10 | 1.7 | ND | |
| 148 | SATYA | 22 | 21744 | primi | B | 34 | VB+P+T | + | HT,A | 1 | M | ceph | | CS | 3 | 150gms | 5/10,7/10 | 2.8 | | |
| 149 | THENMOZHI | 31 | 22392 | G2P1L1 | B | 35 | VB+P+T | + | HT,A | 1 | R | ceph | | CS | 4 | 200gms | 6/10,8/10 | 2.23 | | |
| 150 | NAZIN | 18 | 24342 | primi | B | 36 | VB+P+T | - | HT,A | 3 | M | ceph | S,CA,PPH | CS-CO | 8 | 650gms | 0/10 | 2 | IUD | |
| 151 | BASHEERUNISA | 35 | 24709 | G4P1L1A2 | B | 34 | VB+P+T | + | A | 2 | M | ceph | | CS | 3.5 | 200gms | 5/10,7/10 | 1.9 | AD | |
| 152 | ADHILAKSMI | 20 | 24905 | primi | B | 34 | VB+P+T | + | HT,A | 2 | M | TR LIE | | CS | 2 | 250gms | 6/10,8/10 | 1.9 | AD | |
| 153 | PREMA | 24 | 25261 | primi | B | 29 | VB+P+T | - | HT,A | 3 | M | ceph | S,CA,PPH | CS-CO | 5.5 | 400gms | 0/10 | 2.5 | IUD | |
| 154 | VIJAYA | 20 | 25744 | primi | B | 35 | VB+P+T | + | HT,A | 1 | M | ceph | | CS | 3 | 250gms | 6/10,7/10 | 1.8 | ND | |
| 155 | ESWARI | 25 | 23698 | G4P1L1A2 | B | 30 | VB | + | HT | | | ceph | | CS | | | | | | |
| 156 | REMADEVI | 26 | 25829 | G2P1L1 | B | 38 | VB | + | HT | | | ceph | | CS | | | | | | |
| 157 | BHUVANA | 25 | 25832 | G2P1L1 | B | 36 | P | + | | 0 | C | ceph | | VAG | | 100gms | 6/10,7/10 | 2.6 | | |
| 158 | RAMYA | 32 | 25840 | G3P2L2 | B | 37 | P | + | A | 0 | C | ceph | | VAG | | 50gms | 6/10,7/10 | 2.8 | | |
| 159 | VIJI | 33 | 25860 | primi | B | 37 | VB+P | + | A | | | ceph | | CS | | | | | | |
| 160 | RAGHAVI | 28 | 25849 | G2A1 | B | 37 | VB | + | A | | | ceph | S,PPH | CS | | | | | | |
| 161 | KAMALA | 25 | 25832 | primi | B | 36 | VB+P | + | | | | ceph | | CS | | | | | | |
| 162 | PRIYA | 27 | 26429 | G2P1L1 | B | 36 | | + | HT,A | | | ceph | S,PPH | CS | | | | | | |
| 163 | VENMATHI | 38 | 26496 | primi | B | 32 | VB+P+T | + | HT,A | 2 | M | ceph | | CS | 2 | 250gms | 5/10,7/10 | 1.48 | | |
| 164 | PRAVEENA | 27 | 26429 | G2P1L1 | B | 34 | VB+P+T | + | A | 2 | M | ceph | | CS | 3 | 300gms | 6/10,8/10 | 1.5 | | |
| 165 | SAAIKALA | 19 | 26431 | primi | UB | 30 | VB+P=T | + | A | 2 | M | ceph | | VAG | 6 | 350gms | 0/10 | 1.3 | SB | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 166 | ARUNA | 22 | 26512 | G2P1L1 | B | 35 | VB | + | A+RE | 2 | M | ceph | | VAG | 5 | 200gms | 4/10,7/10 | 2.2 | | |
| 167 | RENUKA | 32 | 26332 | G3P2L2 | B | 36 | VB | + | HT | | | ceph | | VAG | | | | | | |
| 168 | RANI | 27 | 26351 | G2P1L1 | B | 34 | VB | + | HT | | | BR | | CS | | | | | | |
| 169 | VANAJA | 26 | 26361 | G3P2L1 | B | 35 | VB | + | HT | | | ceph | | VAG | | | | | | |
| 170 | KANMONI | 26 | 26490 | G2A1 | B | 36 | VB | + | | | | ceph | | CS | | | | | | |
| 171 | VENDA | 23 | 26481 | G3P2L2 | UB | 36 | VB+P+T | + | A | 2 | M | ceph | | VAG | 4 | 250gms | 5/10,7/10 | 2.4 | | |
| 172 | AMMU | 24 | 26516 | G2P1L1 | B | 35 | VB | + | A | 2 | M | ceph | | VAG | 3 | 200gms | 5/10,7/10 | 2.2 | | |
| 173 | SHAKILA | 26 | 26520 | G3P1L1A1 | UB | 34 | VB | + | | | | ceph | | CS | | | | | | |
| 174 | RUKKU | 32 | 26528 | G2P1L1 | B | 35 | VB | + | | | | ceph | | CS | | | | | | |
| 175 | BAGLAKSMI | 25 | 26501 | primi | B | 37 | VB | + | | | | ceph | | CS | | | | | | |
| 176 | DEVAGI | 24 | 26534 | primi | B | 36 | VB | + | HT | | | ceph | | CS | | | | | | |
| 177 | PRABHA | 23 | 26540 | primi | B | 36 | VB | + | | | | ceph | | CS | | | | | | |
| 178 | VEENA | 27 | 26531 | primi | B | 37 | VB | + | | | | ceph | | CS | | | | | | |
| 179 | SRIDEVI | 26 | 26539 | G2P1L1 | B | 36 | VB+P | + | | | | BR | | CS | | | | | | |
| 180 | AKILA | 25 | 26541 | G3A2 | B | 35 | VB+P | + | HT | | | ceph | | VAG | | | | | | |
| 181 | PADMA | 23 | 26550 | G2A1 | B | 38 | VB | + | | | | ceph | | CS | | | | | | |
| 182 | SASIREKA | 21 | 26559 | primi | B | 36 | VB | + | | | | ceph | | CS | | | | | | |
| 183 | VIMALA | 26 | 26487 | primi | B | 34 | VB+P+T | + | HT,A | 2 | M | ceph | | CS | 4 | 200gms | 6/10,7/10 | 2.1 | | |
| 184 | AMBUJAM | 24 | 26570 | G2P1L1 | B | 35 | VB+P+T | + | A | 2 | M | ceph | | CS | 3.5 | 150gms | 5/10,7/10 | 2.2 | | |
| 185 | RAJESWARI | | 26556 | G2P1L1 | B | 35 | VB | + | HT | | | ceph | | CS | | | | | | |
| 186 | ELIZABETH | 22 | 31620 | G2P1L1 | B | 32 | VB +P+T | + | HT+TR | 1 | M | ceph | | VAG | 2 | 250gms | 6/10,8/10 | 2.1 | | |
| 187 | SUGUNA | 25 | 31711 | primi | B | 39 | VB+P+T | _+ | HT,A | 2 | M | ceph | | VAG | 3 | 250gms | 6/10,8/10 | 2.6 | | |
| 188 | USHA | 28 | 31690 | G4P2L1A1 | B | 36 | VB+P+T | _ | | 3 | M | ceph | S,CA,PPH | VAG | 2 | 600gms | 2/10,3/10 | 800 | IUD | |
| 189 | RANJANI | 23 | 31710 | G2A1 | B | 35 | P+T | + | | 0 | C | ceph | | VAG | | 100gms | 7/10,8/10 | 2 | | |
| 190 | REKHA | 20 | 31724 | G3P2L2 | B | 36 | VB+P+T | - | HT,A | 3 | M | ceph | S,PPH | VAG | 2 | 400gms | 0/10 | 1.5 | IUD | |
| 191 | DEEPA | 30 | 31740 | G3P1L1A1 | B | 32 | VB+P+T | + | | 1 | M | ceph | | CS | 2.5 | 250gms | 6/10,7/10 | 1.6 | AD | |

KEY TO MASTER CHART

| | | |
|---|---|---|
| Ob.Code | : | Obstetric Code |
| B | : | Booked |
| UB | : | Unbooked |
| G.Age | : | Gestational Age |
| Wks | : | weeks |
| S/S | : | Signs,symptoms |
| FH | : | Fetal heart |
| Risk Fact | : | Risk Factors |
| MAT Compli: | | Maternal complications |
| S | : | Shock |
| CA | : | Coagulation abnormality |
| PPH | : | Postpartum Haemorrhage |
| SC | : | Short cord |
| RE | : | Recurrence |
| RF | : | Renal failure |
| TW | : | Twins |
| M | : | Mixed |
| C | : | Concealed |
| R | : | Revealed |
| TR | : | Trauma |
| HY | : | Hydramnios |
| VB | : | Vaginal bleeding |

| | | |
|---|---|---|
| P | : | Pain abdomen |
| T | : | Tense tender uterus |
| M.O.D | : | Mode of Delivery |
| Ab-Del-Int | : | Abruption delivery interval |
| Ceph. | : | cephalic |
| BR | : | breech |
| TR lie | : | Transverse lie |
| HT | : | Hypertension |
| A | : | Anaemia |
| Wt | : | weight |
| RP clots | : | Retroplacental clots |
| P.O | : | perinatal outcome |
| M.M | : | Maternal mortality |
| IUD | : | Intra uterine fetal death |
| ND | : | Neonatal death |
| AD | : | Admission |
| Cs | : | Caesarean section |
| VAG | : | Vaginal delivery |